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*Meyer, E., and Arnold, L., *Amer. J. Digest. Dis.*, 5:418.

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POST-OPERATIVE INTESTINAL OBSTRUCTION

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Brisbane

THIS article deals with intestinal obstruction which complicates an abdominal operation and which develops while the patient is still in hospital under constant observation. It is an unpleasant fact that such cases often carry a worse prognosis than those developing months or years later. This unhappy state exists because it is not generally realized that early post-operative obstructions are characterized by atypical symptomatology.

The most common mistake by far is to regard these early cases of mechanical obstruction, until they are far advanced, as paralytic ileus. Then, usually it is too late for operation to be successful.

After every abdominal operation there is a period of temporary gut paresis which lasts about forty-eight hours. When peristalsis returns to the bowel, the first movements are inco-ordinated and irregular and give rise to the typical colic of "wind pains". Usually flatus is passed soon after, and the patient rapidly settles down to a satisfactory convalescence.

Sometimes this temporary paresis passes imperceptibly into a true paralytic (or adynamic) ileus. Classically, this is characterized by vomiting and abdominal discomfort, rather than pain. Examination reveals a distended abdomen and absent peristaltic sounds. Before the days of intravenous therapy and intestinal suction, this was frequently fatal. It is now known that the cause of death in such cases is dehydration and electrolyte imbalance due largely to the distended, paralysed and atonic bowel. If the distension is overcome by gastric or intestinal suction and the fluid balance maintained quantitatively and qualitatively, then the bowel will recover its tone in time and the patient will recover without operation.

The difficulty arises in distinguishing these cases of paralytic ileus which will recover without operation from those cases of mechanical obstruction which require operation for their cure.

Here it is proposed to discuss, with particular reference to diagnosis and management, twenty-four cases with which I have been closely associated over the past three years. It is hoped that a critical survey revealing obvious errors in the past will point the way to a more successful management of these cases in the future.

Table 1

Post-operative intestinal obstruction mortality in twenty-four cases

	Cases	Deaths
Mechanical Obstruction	20	6
Paralytic Ileus	4	0

Analysis of Cases

The only cases discussed are those in which the diagnosis has been confirmed either at operation or by autopsy. However, it should be realized that, over this three year period, there have been many cases of typical paralytic ileus and a lesser number of undoubted mechanical obstructions which have subsided on the conservative measures of intravenous therapy and intestinal suction.

Of the twenty-four cases, four were proved to be due to paralytic ileus, and twenty were due to mechanical obstruction (see Table 1). There was a fatal termination in six cases, all of which were mechanically obstructed, and in five of which an earlier operation might have saved the patient. There were no deaths in the four cases of paralytic ileus in which an unnecessary operation was performed. In nine cases the original operation which immediately preceded the obstruction was gynaecological. In three of these, however, the condition was found to be due to paralytic ileus. The distribution has been summarised in Table 2.

TABLE 2

Post-operative intestinal obstruction; operation preceding the obstruction

Gynaecological Procedures	9 cases
Appendicectomy	3 „
Hernial Repair	3 „
Excision of Meckel's Diverticulum	2 „
Division of Adhesions	1 „
Abdomino-perineal Resection	1 „
Suture of Perforated Peptic Ulcer	1 „
Resection of Colon	1 „
Haemoperitoneum ? aetiology	1 „
Cystoscopy	1 „
Gastrotomy	1 „

It is not generally realized that the classical features of mechanical obstruction are often absent in the early post-operative period or are masked by analgesic drugs. Only seven out of twenty cases had the typical symptoms of epigastric colic, vomiting and constipation (see Table 3). In eleven cases of mechanical obstruction, pain was either absent or not sufficient to attract attention, while the patient in one case of paralytic ileus suffered severe intestinal colic. Flatus was passed on repeated occasions in six cases of mechanical obstruction and the bowels moved in two cases after the onset of mechanical obstruction.

TABLE 3

20 cases of post-operative mechanical obstruction

Classical symptoms	7 cases
Atypical symptoms	13 „
Absent (or minimal) pain	11 „
Passage of Flatus	6 „
Passage of Faeces	2 „

TYPICAL CASES

Let us deal initially with the typical cases of early mechanical obstruction following operation. The first sign of trouble occurs after the sixth day when the patient develops severe epigastric colic and vomiting. No flatus or faeces is passed in response to an enema. The abdomen becomes distended (often asymmetrically, and, synchronous with the attacks of colic, turbulent *borborygmi* may be heard. Gentle shaking of the patient with the stethoscope on the abdomen will often elicit a "splash". (It is curious that so few doctors realize that a splash elicited in this fashion is a valuable and early sign of dilated bowel.) If the diagnosis is still in doubt a flat X-ray examination of the abdomen is often of value. Absence of gas in the caecum, together with distended small bowel and fluid levels, will clinch the diagnosis of small bowel obstruction.

Such a case does not pose any problem in management. A duodenal tube is passed and suction commenced. Intravenous therapy is instituted and a close watch kept on the patient. Unless he passes flatus within a few hours, the treatment should be surgical. Laparotomy should be performed and the mechanical obstruction relieved before the distension becomes too great. The author does not believe in the definitive treatment of a post-operative mechanical obstruction by means of an intestinal (for example, Miller-Abbot) tube.

ILLUSTRATIVE CASE

Case 1

Master E.P., aged 14 years, underwent appendicectomy on the 12th June, 1954. The appendix was acutely inflamed with a small local abscess on the side wall of the pelvis. The post-operative course was uneventful till the 22nd June, when he developed colicky abdominal pain and vomiting. His bowels had not opened in the previous two days, and he had not passed flatus for twenty-four hours. X-ray examination of the abdomen was interpreted as showing small bowel obstruction. Duodenal suction and intravenous therapy were commenced immediately prior to laparotomy. That afternoon, his abdomen was re-opened and a mechanical obstruction of the ileum was found. This was due to an adhesion of the lower ileum to the site of the previous abscess. The adhesion was divided and the abdomen closed. His post-operative convalescence was uneventful.

Comment

The small bowel proximal to the obstruction was grossly distended and oedematous, despite

early operation. If operation had been delayed, there is a definite possibility that perforation would have occurred (Johnson 1955).

Of the seven cases with typical symptoms two had multiple obstructions. One of these cases required three, and the other four, operations *in toto* before recovery.

ATYPICAL CASES

There were thirteen cases of mechanical obstruction with atypical symptoms. Of these, six had a fatal outcome due largely to late diagnosis.

Paralytic ileus was the main source of confusion and the author believes that in some cases it was the correct initial diagnosis. However, the problem becomes complicated because paralytic ileus, when recovering, may merge imperceptibly into a mechanical obstruction. It should be realized that vomiting and distension which recurs (even in the absence of pain) after a patient has apparently recovered from paralytic ileus are most likely due to a mechanical obstruction and rarely due to a relapse of the paralytic ileus.

The following case illustrates the difficulty of diagnosis.

Case 2

Mrs. P.R., aged 39, was operated upon on the 16th September, 1952, for a ruptured right ectopic gestation, the right Fallopian tube being excised. On the second post-operative day, she looked sick, began to vomit, and the abdomen was distended. Paralytic ileus was diagnosed since she had passed no flatus and peristaltic sounds were absent. A duodenal tube was passed and aspirations begun. On the next day, although she had passed some flatus, the aspirations were larger and intravenous therapy was begun. On the 20th September, she was noticed to be dehydrated and distended although she was still passing flatus. Her general condition improved over the next couple of days, her bowels opening several times on the 22nd September. However, the distension remained and a "splash" was present.

On the 23rd September, there was serious deterioration in the general condition. Pain appeared for the first time, associated with diarrhoea, distension and increasing aspirations. The diagnosis was still considered to be paralytic ileus and vigorous resuscitation was carried out. She improved greatly over the next two days, by which time she denied any pain and said she was hungry.

On the 26th September, she suffered a sudden collapse with peripheral circulatory failure. The pulse rate was 130 per minute, and the blood pressure 85 systolic and 60 diastolic mm. Hg. She was confused, irrational and obviously very ill. She began vomiting faeculent fluid and her bowls opened once during the day. Her abdomen was distended and soft and bowel sounds could be heard. She was thought to be suffering from a biochemical upset, so intravenous therapy was ordered to restore the serum electrolyte levels to normal. By the next day, she had improved slightly, although her urinary output was low. X-ray examination of the abdomen showed distended small bowel but no fluid levels and some gas in the colon. She was now believed, in spite of the lack of pain, to have mechanical small obstruction.

Laparotomy was performed that afternoon (27th September) and revealed advanced mechanical small bowel obstruction due to an adhesive band constricting the ileum one inch from the caecum. The band was divided and the patient recovered after a somewhat stormy convalescence.

Comment

This patient developed paralytic ileus which improved after a few days and her bowels opened. Then vomiting and distension recurred. The lack of pain and profound constitutional disturbance suggested a relapse of paralytic ileus with biochemical upset. However, almost certainly she was mechanically obstructed at this stage and should have been operated upon four days earlier than she was.

FATAL CASES

The six fatal cases will be discussed in detail as each has a lesson to teach. They were all symptomatically atypical and this caused delay in diagnosis and operation. It is realized that, in the terminal phases of a typical mechanical obstruction, pain is absent due to the gross distension and atony of the bowel. However, in most of these cases, pain was absent or quite minimal in the early stages.

Case 3

L.F., male, aged 49, was admitted on the 12th July, 1954, with a perforated duodenal ulcer which was treated by immediate operative suture. His progress was satisfactory until the 20th July when he developed cramping lower abdominal pain. He was found to have a large pelvic abscess from which twenty ounces of foul pus was drained by the rectal approach that day. However, the cramping abdominal pain persisted and, on the 22nd July, although he was passing wind, he was quite distended and a "splash" was elicited. Flat X-ray examination of the abdomen revealed small bowel distension with fluid levels and, as he was not appreciably improved by the next day, it was decided to operate. Laparotomy revealed advanced small bowel obstruction due to adhesion of the ileum in many places to the site of the pelvic abscess. During the freeing of the adhesions, the small bowel was perforated in two places and these holes were closed in two layers.

The patient developed a small bowel fistula on the 29th July. This was treated conservatively until the patient became grossly dehydrated from it, when an attempt was made to close it on the 6th August. However, this was unsuccessful and he finally died on 30th August. Autopsy revealed cachexia and cerebral oedema.

Comment

When drainage per rectum of the pelvic abscess did not relieve the obstruction, laparotomy should have been performed the next day. The combination of infection plus obstruction is particularly dangerous and demands early surgery.

Case 4

H.H., female, aged 58, was admitted on the 8th June, 1953, with acute abdominal pain which was thought to be due to acute appendicitis. The abdomen was opened through a McBurney incision, but the appendix was not inflamed. When a limited exploration revealed no other cause for the pain, the appendix was removed. Her condition was considered satisfactory for the first two post-operative days, although she was noticed to have abdominal distension. On the 11th June, she vomited copious quantities of faeculent fluid and was grossly distended. She complained of no abdominal pain and was only mildly tender in the lower abdomen. Her general condition was poor, as evidenced by a blood pressure of 70 mm. Hg. systolic and 50 mm. diastolic. An enema produced much flatus but no faeces. An X-ray revealed dilated loops of small bowel and an examination of the blood showed the presence of 15,300 leucocytes per cubic millimetre. She was considered to have peritonitis and was treated by duodenal suction and intravenous therapy. Next day, she was improved and now a mass was palpable with overlying tenderness and guarding in the left iliac fossa. Her condition remained unchanged during the next three days—she complained of no pain, passed flatus but the abdominal distension remained.

On the 17th June she became obviously ill again. She began vomiting, the abdomen was still distended and the bowel sounds were diminished. X-ray examination of the abdomen showed distended small bowel with fluid levels. By now, she was thought to be a mechanical obstruction perhaps from resolving peritonitis and it was decided to treat her by intestinal intubation. A Miller-Abbot tube was passed and an X-ray next day showed it to be at the duodeno-jejunal flexure. The tube was well down the small intestine on the 19th June but, in spite of the fact that she stated she was passing flatus, her condition was still poor. In view of the failure of intestinal intubation, operation was performed. She was found to have a closed loop small bowel obstruction due to ill-defined adhesions in the left iliac fossa.

The bowel which was gangrenous was perforated during delivery, and the only treatment possible was resection and exteriorisation of the ileum. She died six hours later. Permission for autopsy was refused.

Comment

Delay in diagnosis of mechanical obstruction was due to almost complete absence of pain and the repeated passage of flatus. Strangulation should have been suspected from presence of an indefinite mass with overlying tenderness and guarding in the left iliac fossa. Increasing distension and foul aspirations should have suggested earlier operation but the condition was thought to be peritonitis.

Case 5

E.R., male, aged 65, was admitted for urological investigation. Cystoscopy revealed residual prostatic obstruction (in spite of a previous prostatectomy) and a urethral stricture. Sounds were passed on 28th January, 1953, to relieve the stricture. On the 30th January he developed mild colicky abdominal pain, and next day began vomiting. His bowels had not moved for six days, and he had not passed flatus for two days. There was a good result from an enema. The vomiting was relieved by the passage of a duodenal tube. On the 1st February, he complained of a minimum of pain only but was greatly distended, bowel contours being visible. He had passed no flatus, and bowel sounds were present but not increased. A "splash" could be elicited with ease. Intravenous therapy was ordered and he was much better next day, although very distended. X-ray examination of the abdomen was interpreted as large bowel distension with fluid levels present. Blood urea was 194 milligrams per cent. In spite of the lack of pain, operation was advised to relieve the presumed mechanical obstruction. Laparotomy on 2nd February revealed advanced mechanical small bowel obstruction due to adhesions between the terminal ileum and the anterior abdominal wall in the region of the previous prostatectomy. The bowel was so distended that intra-mural gangrene had occurred in parts. During the division of the adhesions, the bowel wall was accidentally perforated with a spill of faeculent fluid into the peritoneal cavity. The perforation was oversewn, but the abdomen could not be closed without an ileostomy to provide decompression. The patient failed to rally and died on 4th February. Faeculent peritonitis was found at autopsy.

Comment

It is incredible that the bowel at operation should have been almost gangrenous from distension and yet the patient suffer an absolute minimum of pain. For two days preceding the second operation, he had virtually no pain at all. Operation was delayed on this account because he was considered to have paralytic ileus and uraemia.

Case 6

Mrs. N.H., aged 28, was admitted on 17th April, 1955, with typical clinical features of intestinal obstruction. In addition, she was known to be three months' pregnant. Urgent laparotomy was performed and revealed mechanical small bowel obstruction due

to a fibrous band in the ileo-caecal region. The band was divided and the abdomen closed. Next day, she miscarried and required a curettage. Duodenal suction and intravenous therapy were continued and on 20th April, she denied any pain but the abdomen was still distended. She passed flatus next morning but, in view of the distension and aspirations, intravenous therapy and suction were continued. On 22nd April, the distension was less and she passed more flatus. However, on 23rd April, the abdomen became grossly distended and a loud "splash" could be heard on shaking the patient. Rectal examination revealed a tender mass in the Pouch of Douglas. X-ray examination of the abdomen showed distended small bowel. A provisional diagnosis of pelvic abscess causing obstruction was made, particularly as she had been running an intermittent temperature. A low spinal anaesthetic was given in order to drain the abscess per rectum and while on the operating table, the patient passed liquid faeces. When curved forceps were pushed through the rectal mucosa, there was a loud 'pop' and the patient immediately complained of general abdominal pain. It was suspected that the bowel had been perforated, so laparotomy was immediately performed. A loop of ileum which was grossly distended and the site of intramural gangrene had been perforated causing faecal peritonitis. The small bowel in the ileocaecal region was quite collapsed but the patient's condition did not allow of further exploration. During the operation, the bowel was holed again. This was oversewn and an ileostomy performed at the site of the original perforation. The patient, despite vigorous resuscitation, died twelve hours later. Autopsy showed faecal peritonitis and a mechanical small bowel obstruction due to an adhesion extending from the site of an earlier appendicectomy to the round ligament.

Comment

The following comments seem pertinent:

1. She complained of virtually no abdominal pain after her first operation.

2. She passed flatus intermittently and her bowels actually opened on the operating table, even though she was completely obstructed.

3. The pelvic collection was diagnosed in error. The mass palpable was a tender retroverted post-abortal uterus.

4. Almost certainly the original obstruction was caused by multiple bands, not all of which were divided at the original operation. The ones remaining caused her subsequent obstruction.

Case 7

Mrs. O.C., aged 53, was admitted to hospital for investigation of chronic pain in the left iliac fossa. It was thought to be due to disease of the left ovary and abdominal section was performed on the 4th November, 1953. The left ovary was removed and many adhesions were noted to be dragging the uterus over to the left side of the pelvis. Next day, she complained of mild hypogastric pain only and the pulse rate was 76 beats per minute. On the second post-operative day, at 9.0 a.m., she began vomiting copiously. Examination revealed abdominal distension with a "splash" and absent bowel sounds. She made no complaint of any pain. A provisional diagnosis of paralytic ileus was made, and duodenal suction and intravenous therapy commenced. At 12.30 p.m., her blood pressure was systolic 80, diastolic 60 m.m. Hg.

By 2.30 p.m. she was desperately ill with the blood pressure not recordable. There was general tenderness and regidity in the distended abdomen. At this stage, mechanical obstruction was considered as a possible diagnosis but she was too sick for surgery. At 10.30 p.m. she complained of pain in the back and the hypogastrium. She died soon after, in irreversible shock. At autopsy she was found to have a strangulating small bowel obstruction, due to an adhesion between the ileum and anterior abdominal wall just to the left of the laparotomy incision.

Comment

This patient died within two days of operation from a strangulating small bowel obstruction. The rapid and gross deterioration in her condition suggested such a catastrophe but she was considered too sick for operation.

Case 8

Mrs. N.W., aged 67, complained of persistent upper abdominal pain which was thought to be due to a gastric ulcer. Gastrectomy was contemplated but, at operation on the 12th October, 1953, no ulcer could be found on external examination of the stomach. The stomach was opened, and a small healing gastric ulcer was found. It was now considered unjustified to carry out a resection, so the stomach was closed in two layers and the abdomen closed without any further procedure. On the third post-operative day, she was distended and mentally confused. Next day she began vomiting. On 18th October, her abdomen was still distended and a mass was now palpable in the right iliac fossa. She complained of no pain other than in the wound. There was no response to two enemata. Visible peristalsis was present and, in view of the X-ray findings of dilated small bowel, she was considered to have a mechanical obstruction.

Laparotomy was performed on 18th October and complete small bowel obstruction due to a volvulus of the caecum was found. The caecum was viable, so the bowel was untwisted and caecopexy performed. Her post-operative course was stormy. Over the next two weeks she was intermittently obstructed and required intravenous therapy and enemata. Because she was considered to have a recurrence of the volvulus, it was decided to re-open the abdomen on 16th November. This disclosed a condition of plastic peritonitis with multiple adhesions in the right iliac fossa. During the exploration, the small bowel was accidentally opened and later oversewn. Caecostomy was then performed. After the operation, an intestinal tube was passed but this became stuck at the duodeno-jejunal flexure. In spite of supportive measures, she died on the 22nd November, permission for autopsy being refused.

Comment

The ending may have been happier had caecostomy been done at the second operation. The third operation was ineffective. Ileo-transverse anastomosis may have been a better procedure, especially if done earlier.

CASES OF PARALYTIC ILEUS MISDIAGNOSED AS MECHANICAL OBSTRUCTION

There were four of these cases which came to laparotomy.

Three cases followed hysterectomy (2 with a small haemoperitoneum) and one followed repair of an epigastric hernia. In one of them, pain was of considerable severity.

Case 9

Mrs. A.G., aged 34, underwent total hysterectomy for menorrhagia on 5th October, 1954. Her condition did not cause concern until 8th October when she complained of spasms of colic coincident with turbulent *borborygmi*. The abdomen was tense, tender and slightly distended. There was definite muscle guarding with rebound phenomena. X-ray examination of the abdomen showed distended bowel (? small, ? large) but no fluid levels. As an early strangulating obstruction could not be excluded, laparotomy was performed. A small amount of old blood clot was found but no active bleeding site. There was uniform distension of the small and large intestine typical of paralytic ileus. The abdomen was closed and the patient made an uneventful recovery.

Comment

Haemoperitoneum was not considered as a possible diagnosis because the patient was a good colour. The undoubted small bowel colic can only be explained as an attempt by Nature to overcome the paralytic distension of the large bowel. The interpretation of the X-ray was wrong in that the distended loops were actually colon.

In the other three cases, pain was not marked but persistant vomiting and distension justified laparotomy. The following case history gives a good illustration.

Case 10

Mrs. H.Q., aged 50, was an obese lady who underwent total hysterectomy and bilateral salpingo-oophorectomy for fibroids on 23rd February, 1954. During the next two days, she began vomiting and complained of mild abdominal pain. Peristaltic sounds were present and a provisional diagnosis of paralytic ileus was made. On 27th February, she complained of some pain in the left upper quadrant of the abdomen and was noticeably distended. An X-ray of the abdomen showed distended small bowel and gas in the large bowel. She was still considered to be suffering from paralytic ileus and now an intestinal tube was passed into the duodenum under the radiological screen. Intravenous therapy was continued.

On the 28th February, she denied any pain, and passed flatus even though the abdomen was distended. Next day, she seemed worse — the aspirations were faeculent, and the bowel sounds were increased and tinkling. It was thought she was passing into a mechanical obstruction. On 1st March she had a bowel action but her abdomen was still distended and tight. Next day, she passed flatus two or three times but looked very ill, with gross abdominal distension and large quantities of faeculent material were aspirated. X-ray examination of the abdomen was interpreted as showing small bowel distension with some gas in the colon. It was thought she had a partial mechanical obstruction, so laparotomy was performed on 2nd March. This showed gross distension of small and large bowel with an enormous caecum and was typical of paralytic ileus. The abdomen was closed after exploration and she recovered after a relatively smooth convalescence.

Comment

This was a most difficult case to diagnose. The paralytic ileus persisted so long that it was thought to have become complicated by mechanical obstruction. In addition, the X-rays were of poor quality due to the obesity and were probably misinterpreted. However, laparotomy was not harmful.

DISCUSSION

Let us examine in some detail the symptomatology in an effort to distinguish the mechanical from the adynamic obstruction.

(a) *Pain.* When pain occurs in post-operative cases, close questioning is often necessary to elicit that the pain is colicky in nature and not associated with the recent wound. Usually epigastric colic is due to a mechanical block but occasionally it may occur in paralytic ileus.

However, it should be emphasized that in at least 50 per cent. of cases, mechanical obstruction develops or complicates paralytic ileus without the patient being aware of any pain worse than abdominal discomfort.

(b) *Vomiting.* If the vomitus is large in amount, brown or evil-smelling, and this persists more than a short while in spite of routine conservative measures, mechanical obstruction should be suspected. Likewise, a recurrence of vomiting after it has once subsided is suspicious of mechanical obstruction. The colour and smell of the fluid vomited or aspirated is quite as important as the amount.

(c) *Constipation.* In paralytic ileus, there has often been no bowel action since operation, whilst in mechanical obstruction there may well have been one before the onset of symptoms. Often faeces and flatus will be passed after the onset of a mechanical obstruction, as the bowel distal to the obstruction is still able to evacuate its contents. However, the passage of large amounts of gas with coincident relief of distension usually indicates at least partial release of the obstruction.

(d) *Distension.* Distension persisting for some days post-operatively is of serious significance even in the absence of pain. Mechanical obstruction should be considered should distension recur after it has once subsided. Note that cases of small bowel obstruction occurring within a week or so of an operation seem to become much more distended than cases occurring months or years later.

(e) *Constitutional signs.* Persistent tachycardia accompanied by distension is a bad sign and should not be allowed to continue for long without consideration of operation. Even though critically ill, the mechanically obstructed patient is often surprisingly alert, while the patient with paralytic ileus is often dull and apathetic.

(f) *Peristalsis.* Audible and visible peristalsis has doubtful significance and too much reliance should not be placed on these signs. The author has operated on a case of paralytic ileus with audible tinkling and visible waves of peristalsis and, on many occasions, has seen cases of mechanical obstruction with absent bowel sounds.

These confusing signs may be explained in the following manner. Visible and audible peristalsis may occur in loops of intestine in an attempt to overcome localised paralytic ileus, while peristaltic sounds may be absent in the atonic bowel of an advanced obstruction.

(g) *Strangulation.* This is characterized by severe colic, tenderness and rigidity. It is usually readily apparent from the severe constitutional signs and collapse that a catastrophe has occurred. Curiously enough, however, strangulation is not common in the early post-operative phase.

(h) *Radiological signs.* These are notoriously difficult to interpret in the early post-operative phase, as signs of mechanical obstruction often overlap those of paralytic ileus. However, X-rays may be a most valuable aid and should be taken in the Department, as portable films are usually of such poor quality as to be valueless. Views of the abdomen should be taken in the supine, erect and the left lateral recumbent positions. The latter is especially suitable for very sick patients in whom the erect film is impossible.

FIG. I. Flat X-ray of the abdomen taken with patient erect. This is a case of mechanical small bowel obstruction following appendicectomy. Note distended loops of small bowel, the presence of fluid levels, and the absence of gas in the large bowel.

Mechanical small bowel obstruction may be diagnosed on the following signs (Fig. 1):

1. Presence of distended loops of small bowel with fluid levels present in the upright and lateral films.

2. Absence of gas in the large bowel. If an enema has been given recently, gas may be present in the colon but the large bowel is not distended.

Post-operative large bowel obstruction is uncommon, and it is practically impossible to differentiate it radiologically from paralytic ileus.

In paralytic ileus, both small and large bowel are grossly distended (Fig. II) and fluid levels may be present in both. In the erect film, the lower abdomen often appears rather opaque due to the presence of free fluid.

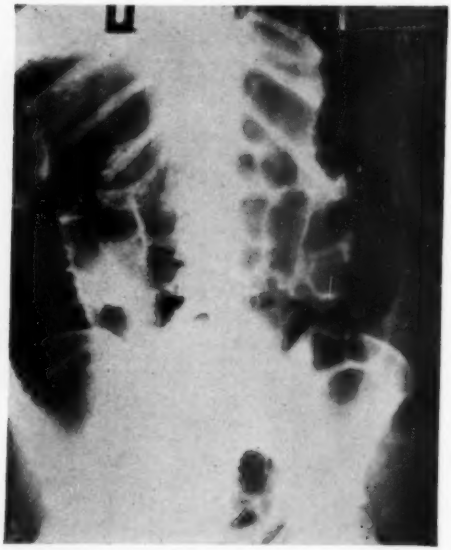

FIG. II. Flat X-ray of the abdomen taken in erect view. This is a case of paralytic ileus following salpingectomy. Note that both small and large bowel are distended.

It should be realized that there are no pathognomonic radiological signs of strangulation.

MANAGEMENT OF THE "POST-OPERATIVE ABDOMEN"

If a patient becomes distended after an abdominal operation, a tube should be passed immediately into the stomach so that the regurgitated contents may be regularly aspirated. Simultaneously, intravenous therapy should be instituted with due regard to the amount and nature of the fluid given.

If (a) there is pain other than in the wound

(b) aspirations remain faeculent, or vomiting recurs when a duodenal tube is removed

(c) distension progresses, or recurs when it has once subsided

(d) there is persistent tachycardia

then the case should be considered as a possible mechanical obstruction. If the signs persist for more than a short time without relenting, then operation should be undertaken.

The author believes it is much less dangerous to operate on a relatively fit patient in the early stages of paralytic ileus than to delay operation in a case of mechanical obstruction. In other words, it is safer to adopt a policy of "look and see" rather than "wait and see".

Intestinal suction using a Miller-Abbot type of tube is no longer favoured by the author as a definitive form of treatment for an established mechanical obstruction. It is still extremely difficult to persuade the tube to pass the pylorus and even more galling for the suction to be ineffective (as it so often is) when once the tube is in the small intestine. Wangensteen (1950) is becoming "more conservative concerning the continued employment of suction when the distension does not relent promptly". The objections to the use of inlying gastro-intestinal tubes to achieve relief of the obstruction without operation may be stated as follows:

(a) the method is not always effective

(b) it is frequently slow in achieving satisfactory decompression, threatening the safety of the patient and disturbing the equanimity of the surgeon.

(c) it may be inadvertently applied to patients with strangulating obstruction in whom immediate operation is always mandatory.

However, a recent trial of intestinal intubation, using the Grafton Smith stylet (see Fig. III), has shown its value as an immediate preoperative manoeuvre. Under the radiological screen, a tube may be guided with the stylet into the upper jejunum, in many cases within half an hour. Then, if operation follows immediately the tube may be guided down the small intestine to the site of the obstruction when the abdomen has been opened. In this way, operative aseptic decompression can be provided and the fatal effects of spillage avoided. It is much safer to divide the offending adhesion when the proximal bowel has been collapsed by the tube than when unruly loops of bowel obstruct the exposure. Unfortunately, lack of time and radiological fa-

ities in a busy institution prevents this method from having universal application. However, the following case illustrates its use.

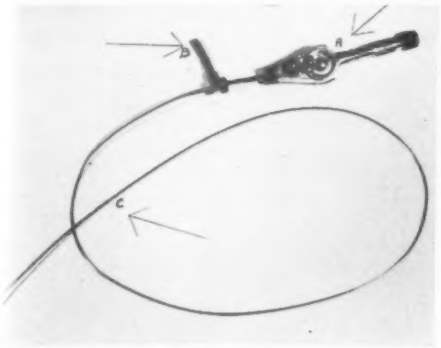

FIG. III. Grafton Smith Flexible Stylet with controllable tip.
(A) Handle incorporating director lever, tension sleeve, and stiffening lever.
(B) Air adaptor for introducing air into stomach of patient during passage of tube.
(C) Controllable tip.

Case 11

R.S., male, aged 75, underwent abdomino-perineal resection for carcinoma of the rectum on 12th November, 1954. His progress was satisfactory until 29th November when he developed typical symptoms of small bowel obstruction, confirmed by X-ray examination of the abdomen. An intestinal tube was passed under the radiological screen using the Grafton Smith stylet. The duodenum was intubated within fifteen minutes. Soon after, laparotomy was performed. Two loops of ileum had become adherent to the suture line of the pelvic floor, causing mechanical small bowel obstruction. The distended proximal bowel was emptied on the table through the intestinal tube prior to the division of the adhesions.

Intestinal suction was continued for three days post-operatively, after which the patient settled down to an uninterrupted recovery.

Comment

Decompression of the proximal bowel at the time of operation made the operation much easier.

Even if the bowel has been decompressed, and the greatest gentleness exercised, the bowel may be perforated in these cases. The reason is that the adhesive band may cut so deeply into the bowel that the entire circumference save the mesentery may be divided, the edges of the gut being held together only loosely by fibrin.

If time permits, the wound should be closed in layers using non-absorbable sutures. Every measure should be taken to prevent later wound disruption, which is a grave complication.

CONCLUSIONS

Early post-operative intestinal obstruction is a calamity for the patient and an embarrassment for the surgeon. The outlook for the patient will be improved when it becomes generally known that the clinical features are atypical in a majority of cases. The surgeon may be saved much anxiety if, in a difficult case, he calls in a consultant who has not been confused by the gradual transition of symptoms from a paralytic ileus into a mechanical obstruction.

SUMMARY

1. Twenty-four cases of early post-operative intestinal obstruction are discussed. Of these, four were due to paralytic ileus, and twenty to mechanical obstruction.

2. There was a fatal termination in six cases, all of which were mechanically obstructed.

3. Paralytic ileus caused delay and confusion in diagnosis.

4. The classical features of mechanical obstruction are often absent in the early post-operative period. In eleven cases, pain was either absent or insufficient to attract attention.

5. Vomiting and distension which recurs after it has once subsided is usually due to a mechanical obstruction and not to a relapse of paralytic ileus.

6. Good X-rays are of great importance in the differential diagnosis between mechanical obstruction and paralytic ileus.

7. Intestinal intubation using the Grafton-Smith stylet is of value as an immediate pre-operative manoeuvre.

ACKNOWLEDGEMENT

I wish to thank Professor Neville Sutton for advice and secretarial assistance during the preparation of this paper, and Dr. A. D. D. Pye, General Superintendent of the Brisbane Hospital, for access to the records.

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NASOPHARYNGEAL CARCINOMA

AN ACCOUNT OF THE CRANIAL NERVE LESIONS FOUND IN 185 CASES

By MARGERY LAWLEY

Department of Surgery, University of Malaya

THE occurrence of cranial nerve lesions in nasopharyngeal carcinoma has become, in recent years, a well-recognized feature of the disease. The first detailed account of these lesions was given by Woltman in 1922 and he was able to collect descriptions from the literature of 44 cases of malignant disease of the nasopharynx with neurological damage, the earliest report having been written in 1858 (Woltman).

Digby (1941) gave a full description of the symptoms and signs of cranial nerve damage in nasopharyngeal carcinoma among the Chinese in Hong Kong but did not specify the incidence of this feature nor the order of frequency in which the different nerves were involved.

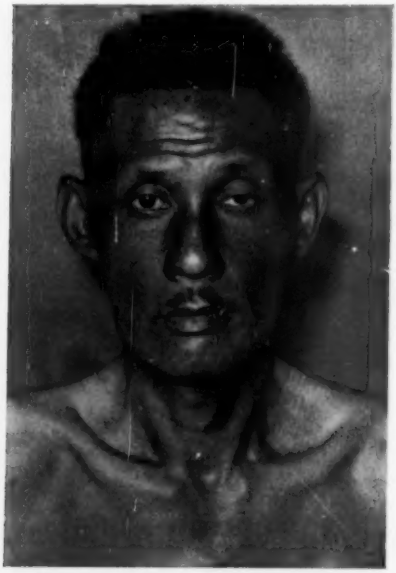

FIG. I. Photograph of a patient showing left-sided ptosis and lateral strabismus.

Godtfredsen (1947) collected 454 cases of malignant disease of the nasopharynx from the four Scandinavian Radiological Clinics and found that there were ophthalmo-neurological manifestations in 38 per cent. He emphasized that these lesions occurred early in the disease and produced figures to show that the large majority of these lesions had developed 4 months before diagnosis was reached.

Simmons and Ariel (1949) in Illinois found that 29.3 per cent. of their 150 cases had cranial nerve lesions on admission to hospital and that a further 7.3 per cent. of patients developed these later.

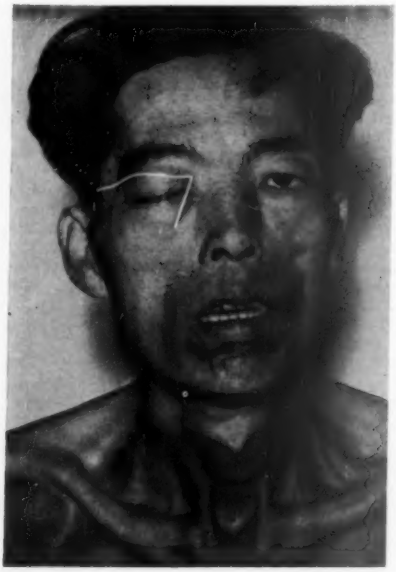

FIG. II. Gross right-sided ptosis.

Godtfredson (1947) found that the trigeminal nerve was involved first and most frequently, followed by the abducens nerve. Woltman (1922) quoted the same two nerves but in reversed order of frequency. Jefferson (1953) found the fifth nerve to be most commonly involved while Simmons and Ariel

(1949) quote the order as sixth, twelfth, fifth. All authorities are in agreement that the olfactory and auditory nerves are seldom involved but that anosmia is due to nasal obstruction and deafness to blockage of the Eustachian tube.

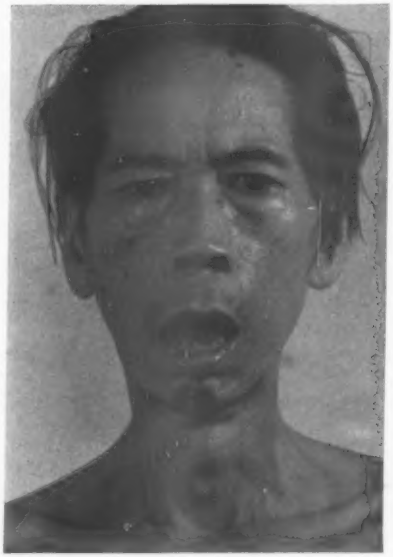

FIG. III. Wasting of right masseter muscle with slight deviation of mandible to side of lesion.

Godtfredsen thought that the nerves were involved by extension of tumour through the basal foramina or by osseous perforation of the bones of the base of the skull to involve the nerves in the cavernous sinus and with possible spread forwards to involve the optic nerve. Alternately the tumour might grow out from the lateral nasopharyngeal wall into the parapharyngeal space to involve the nerves in the jugular foramen and hypoglossal canal. Jefferson described the route of invasion of the nerves in the cavernous sinus as being up the carotid sheath of foramen lacerum then to trigeminal ganglion and so to cavernous sinus and explained the early involvement of the fifth nerve on this basis. In describing the anatomy of the cavernous sinus he points out that the oculomotor nerve enters the sinus through an open dural sleeve half-way along the superior surface and may therefore be spared in posterior lesions of the sinus.

FIG. IV. Bilateral wasting of masseter and temporalis muscles more obvious on the right side. Also bilateral abducens lesions.

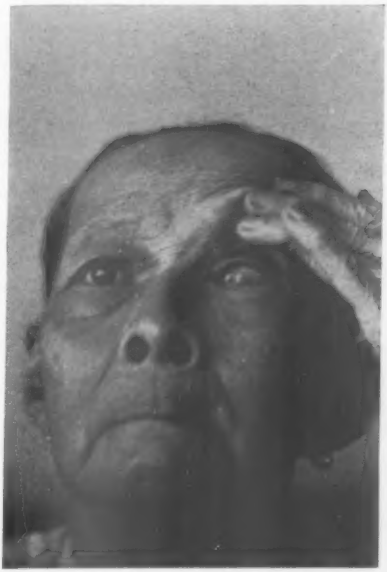

FIG. V. Keratitis of left eye due to lesion of ophthalmic nerve.

Trotter (1911) described how the inferior division of the fifth nerve may be involved by direct outward growth of tumours of the lateral wall.

The ninth, tenth, eleventh and twelfth nerves may be injured in the neck by lymph node metastases as may the cervical sympathetic but Woltman (1922) thought spread more commonly occurred along the base of the skull to involve nerves at jugular foramen or anterior condylar canal.

due to damage of this nerve such as disturbances of lacrimation should be an early symptom of the disease. Digby *et alii* (1941) described a patient who cried frequently; but tears flowed from one eye only, the other remaining dry. Flatman considers that the apparent rarity of involvement of this nerve is due to the fact that, in the past, its significance has not been realized.

Several neurological syndromes have been used in describing the cranial nerve lesions

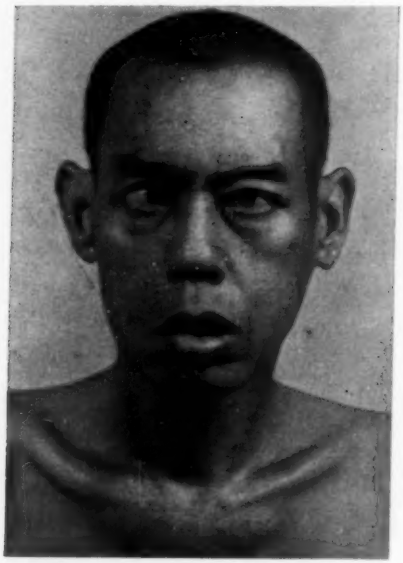

FIG. VIa. Bilateral abducens paralysis with internal strabismus of both eyes. Looking to left.

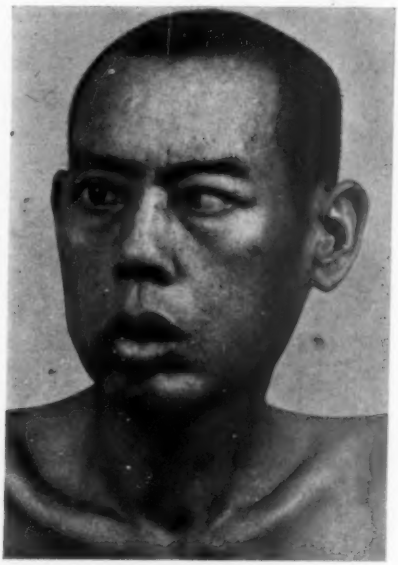

FIG. VIb. Bilateral abducens paralysis with internal strabismus of both eyes. Looking to right.

Mekie (1949) described a case where tumour had penetrated the occipito-atlantoid ligament and spread into the extradural space and thus involved the cranial nerves.

Burger (1934) has shown that the ninth, tenth and twelfth sympathetic nerves may be involved in the parapharyngeal space, the space enclosed by the constrictor muscles medially, the transverse processes of upper cervical vertebrae posteriorly and the ascending ramus of mandible and parotid gland laterally.

Flatman (1954) points out that the nerve of the pterygoid canal is the nerve nearest to the fossa of Rosenmüller and that symptoms

in nasopharyngeal carcinoma. Burger (1934) has emphasized the weakness of classifying the lesions by such a method as one syndrome may progress to another in a short period of time due to the involvement of further nerves.

These syndromes may be summarized briefly as follows:

1. Jacod's (1934) petrosphenoidal syndrome where there is unilateral neuralgia of trigeminal nerve and total unilateral ophthalmoplegia due to involvement of second, third, fourth and sixth nerves.

2. Gradenigo's (1907) syndrome due to lesions at the tip of the petrous temporal bone causing external ocular palsy (usually sixth nerve) with pain in the distribution of the fifth.

3. Godtfredsen (1947) described a syndrome consisting of ophthalmoplegia, usually of the sixth nerve, and a lesion of the twelfth which he claimed was pathognomonic of nasopharyngeal tumours, the sixth nerve lesion being due to an intracranial lesion and the twelfth nerve being involved by cervical metastases.

FIG. VII. Right facial paralysis.

4. The syndrome of the jugular foramen where the ninth, tenth and eleventh nerves are involved.

5. Syndrome of retroparotidean space giving lesions of the ninth, tenth, eleventh, twelfth and cervical sympathetic nerves.

6. Collet (1929) described a syndrome similar to (5) but where the sympathetic chain escaped.

7. The syndrome of Hughlings Jackson (1886) where the tenth, eleventh and twelfth nerves are involved.

8. The syndrome of the parapharyngeal space which has already been described in which there are lesions of the ninth, tenth and twelfth nerves.

Probably the most valuable contribution is that of Jefferson who points out that unilateral nerve palsies or inconsequent palsies of several cranial nerves are strongly suspicious of nasopharyngeal tumours.

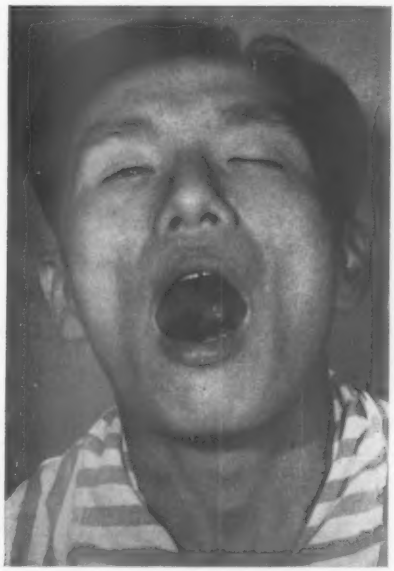

FIG. VIII. Same patient as shown in Fig. IX illustrating paralysis of right half of soft palate.

THE CRANIAL NERVES

The clinical manifestations of lesions of the various cranial nerves are well known and illustrations of these are shown in Figs. I-XII. There are certain points which require elaboration.

Innervation of the soft palate

This is a subject over which there is some disagreement. The palate is supplied by the vagus nerve, probably through fibres derived from the accessory nerve. For the purpose of this study paralysis of the palate is included among vagal lesions. It may also be paralysed by direct pressure of tumour from above or by infiltration of the *M. levator*

FIG. IX. Wasting of left sternomastoid muscle.

palati from tumours of the lateral wall. Trotter's triad of unilateral deafness, paralysis of the soft palate on the same side and an ipsilateral lesion of the mandibular nerve is descriptive of tumours of the lateral wall growing outwards to obstruct the Eustachian tube, to infiltrate the *M. levator palati* and involve the mandibular nerve.

So intimately connected are lesions of the ninth, tenth and eleventh nerves that Walshe (1952) suggests that they be considered as a glossopharyngeal-vagus-accessorius complex. There is much to be said for this both because of the doubt regarding the innervation of the palate and in view of the theory held by some that the superior constrictor is supplied by the ninth nerve. While dysphagia, which is a common late symptom of the disease, may occasionally be due to obstruction by the primary tumour or a lymph node metastasis it seems probable that superior constrictor paralysis is the most common cause.

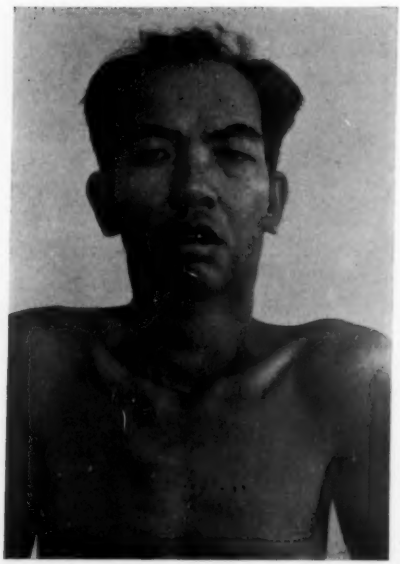

FIG. Xa. Wasting of left trapezius muscle. Seen from the front.

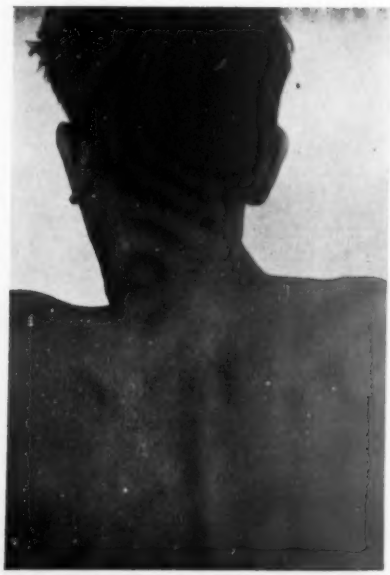

FIG. Xb. Wasting of left trapezius muscle. Seen from the back.

Cranial nerve lesions in 185 cases of nasopharyngeal carcinoma seen in the Civil General Hospital, Singapore, during the years 1947-1955

Of 185 patients suffering from carcinoma of the nasopharynx, 89 (48 per cent.) had cranial nerve lesions, in all but 6 of whom neurological damage was present when they were first seen. Of these 89 cases, 76 per cent. of the lesions were multiple and 25 per cent. bilateral. The average duration of the disease, when the patient presented at hospital, was 13 months — the shortest and longest histories given being 1 month and 5 years respectively. Symptoms referable to the cranial nerves appeared, on the average, 10 months after the onset of disease but in 15 cases occurred as the initial symptom and in one case did not appear until 3½ years after the history began. In 16 cases where there were cranial nerve lesions, no symptoms referable to these were exhibited. The earliest and most common symptom referable to cranial nerve damage was neuralgia or numbness of the face in the distribution of the trigeminal nerve. Diplopia and blurred vision occurred almost as frequently. In one case ptosis and in another dysphagia were the initial symptom. In general, facial paralysis, dysphagia and hoarseness were later features of the disease.

In 7 cases there was complete hemianaesthesia of face and side and front of the head, of which 5 had also a lesion of the sixth nerve. Lesions of the abducens nerve accompanying maxillary and/or ophthalmic nerve involvement were common. The third division and motor branch were only involved together without other branches of the trigeminal in two instances, so that spread through the foramen ovale cannot have been a common occurrence. Direct spread through foramen lacerum to involve maxillary, abducens and ophthalmic nerves would explain the common combination of lesions of these nerves.

Combined lessions of cranial nerves

These fell into the following main groups:

1. Involvement of nerves of the cavernous sinus and of the maxillary nerve (which runs along the inferior border of sinus).

TABLE I

SHOWS THE FREQUENCY WITH WHICH EACH CRANIAL NERVE WAS AFFECTED

Cranial Nerve	*Involved Alone*	*Involved with Other Nerves*	*Total Involved*
I	–	—	—
II	–	14	14
III	1	25	26
IV	–	14	14
V	5	38	43
VI	5	40	45
VII	–	12	12
VIII	–	2	2
IX	–	15	15
X	–	34	34
XI	2	24	26
XII	1	30	31

From this table it is seen that VI and V are most commonly affected, X, XI, XII also frequently as is III.

(a) Complete ophthalmoplegia in 8 cases, accompanied by a lesion of the fifth nerve in 7 of these.

(b) The fifth and sixth nerves together involved in a further 18 cases.

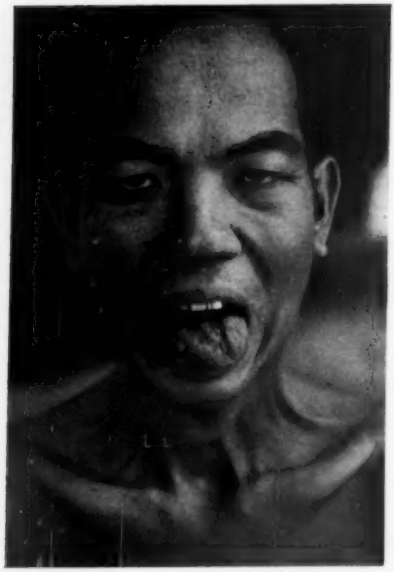

FIG. XI. Same patient as shown in Fig. X showing (left) hypoglossal nerve paralysis.

2. Involvement of the ninth, tenth, eleventh and twelfth nerves and cervical sympathetic chain.

(a) The whole group were involved together in 5 cases—the syndrome of the retroparotidean space, and lesions of the ninth, tenth, eleventh and twelfth nerves without a Horner's syndrome occurred once.

(b) Ninth, tenth and eleventh nerves were involved together in 6 cases—syndrome of the jugular foramen

(c) Tenth, eleventh and twelfth nerve lesions occurred together in 7 instances—Jackson's syndrome.

(d) Ninth, tenth and twelfth nerves were affected together in 3 patients—the syndrome of parapharyngeal space.

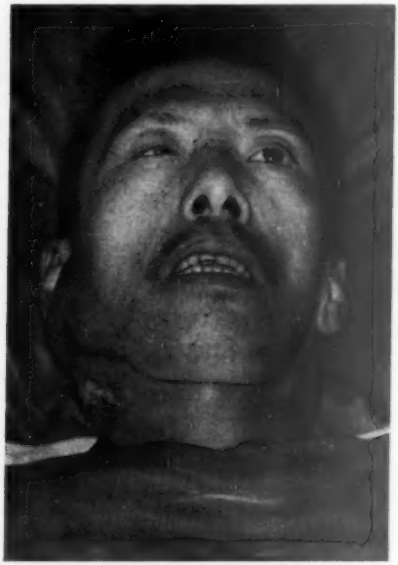

FIG. XII. Horner's syndrome on right side.

TABLE 2

SHOWS THE INCIDENCE OF INVOLVEMENT OF THE VARIOUS BRANCHES OF THE TRIGEMINAL NERVE

Trigeminal Nerve	*Involved Alone*	*Involved with Other Branches of V*	*Involved with Nerves Other Than V*	*Total Involved*
Ophthalmic	–	14	14	16
Maxillary	3	14	3	20
Mandibular	–	14	—	14
Motor	2	17	5	24

3. Godtfredsen's syndrome of involvement of sixth and twelfth nerves together occurred in 4 cases, in three of which the trigeminal was also involved.

Apart from these recognized groupings a wide variety of combinations of cranial nerve lesions, often with widely separated anatomical locations, were found. In only one case was there any evidence of involvement of the nerve of the pterygoid canal.

Radiological findings

In 53 cases (60 per cent.) there was involvement of the base of the skull. This was indicated: (a) by erosion of the middle cranial fossa commonly at the petrous apex in region of foramen lacerum, at foramen ovale or, sometimes, by widespread bone destruction, (b) by destruction of sella turcica or (c) by opacity of the sphenoid sinus. Several cases where the ophthalmic nerve, maxillary nerve and abducens nerve were involved showed no radiological evidence of bone destruction; where the tumour had spread through the foramen lacerum without damaging the cranium.

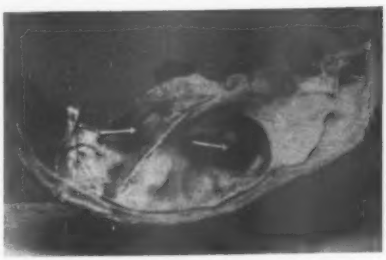

FIG. XIII. Photograph showing tumour deposits in middle and posterior cranial fossae.

DISCUSSION

The incidence of cranial nerve lesions in this series of 185 cases (48 per cent.) is higher than that found by Godtfredsen (38 per cent.) or Simmons and Ariel (36.6 per cent.). The point brought out by Godtfredsen that symptoms referable to cranial nerve damage were an early feature of the disease has been confirmed in this study; that such symptoms appeared on the average 3 months before diagnosis was made emphasizes more than ever the necessity of early recognition of cases of nasopharyngeal carcinoma. In Singapore, where the nasopharynx is the third most common site of cancer, and where such a diagnosis should be in the forefront of every doctor's mind, 45 per cent. of all cases had developed cranial nerve lesions before a correct diagnosis was reached. In some cases the patient did not consult a doctor until the disease was advanced and in 15 cases (8 per cent.) a symptom caused by cranial nerve damage was the first indication of disease.

The high incidence of trigeminal and abducens paralysis found in this series corresponds to that found by other observers. Pain or numbness in the distribution of the fifth nerve was the commonest early symptom and Jeffersen's theory of spread through the foramen lacerum, then to the trigeminal ganglion and thence to the cavernous sinus, appears a reasonable supposition. The optic nerve was never involved without a co-existing lesion of one or more of the nerves in the cavernous sinus and it seems likely therefore that spread continues forward to involve the second cranial nerve.

Lesions of the last four cranial nerves may occur at their exit from the skull, in the parapharyngeal space or in the neck. The frequency of any one syndrome does not necessarily indicate the number of times in which the nerves are involved in a particular location as one or more nerves of a certain group may escape injury.

The widespread and inconsequent palsies found in many cases emphasize the weakness of rigid division of the cranial nerve manifestations of nasopharyngeal carcinoma into separate syndromes.

The facial nerves would appear to be involved at the stylomastoid foramen. If it were injured at the internal auditory meatus one would expect a lesion of the auditory nerve to be present, and whereas facial paresis was present in 12 cases, there was only clinical evidence of the eighth nerve involvement in two cases.

SUMMARY

Forty-eight per cent. of 185 cases of nasopharyngeal carcinoma seen over an eight-year period exhibited evidence of cranial nerve damage. The abducens nerve and trigeminal nerve were the most frequently involved. The lesions fell into two main groups: (a) where one or more of the second, third, fourth, fifth

and sixth nerves were involved, and (b) where there was a lesion of one or more of the last four cranial nerves and of the cervical sympathetic chain.

The early onset of cranial nerve lesions and the consequent need for speedy diagnosis is emphasized.

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PRIMARY CARCINOMA OF THE LIVER

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PAPERS from many parts of the world in recent years which have described primary carcinoma of the liver make it apparent that this uncommon disease is considerably modified by racial and environmental factors. It is rare in European communities but is a major problem among certain native African populations. The aim of this paper is to present an account of the pathology of primary carcinoma of the liver in the State of Victoria and to ascertain whether in this community the disease has developed special characteristics in response to a local environment. Since many of the problems presented by this disease have concerned its association with cirrhosis, particular attention will be paid to cases complicating that condition.

Historical Review

Kelsh and Keiner (1876) described 2 cases of primary carcinoma of the liver and, although as early as 1849 Rokitansky had observed that liver carcinoma was often primary, found mention of only one other case in the literature. Since then many series of cases have been examined and a clear understanding of the morphology and behaviour of groups of these tumours has emerged. Two distinct histological patterns were recognized: one composed of cells resembling parenchymatous liver and the other of epithelium of intrahepatic bile-duct type, for which Goldzieher and von Bokay (1911) coined the terms "carcinoma hepatocellulaire" and "carcinoma cholangiocellulaire" respectively. In the same year Yamagiwa (1911) introduced the subsequently widely used names, "hepatoma" and "cholangioma," in a generic sense to include all tumours, adenomata and carcinomata, composed of and possibly arising from liver cells and bile-duct epithelium respectively. The term "hepatoma" was used, in yet another sense, by some authors who doubted that all apparently malignant liver-cell tumours were carcinomata. They discarded the term "carcinoma" altogether and substituted "hepatoma," implying that their peculiar behaviour was something different from most true cancers (Ewing, 1940).

Racial distribution

The racial differences in incidence of primary carcinoma of the liver were established by Berman (1940) when he found that 37.4 per cent. of all cancers discovered at autopsy among the Bantu people of Africa were primary hepatic carcinoma, contrasted with corresponding figures of 18.7 per cent. for semi-Bantu, 14.1 per cent. for Orientals and 1.1 per cent. for Europeans. From the records of 12,800 consecutive autopsies at Leeds, England, Stewart (1931) found 14 cases of primary carcinoma of the liver—an incidence of 0.11 per cent. In America, Hoyne and Kernohan (1946) collected at the Mayo Clinic 31 cases from 16,303 autopsies (0.19 per cent.) and Strong and Pitts (1933) found similarly, in the white population of Vancouver, 2 cases from 1,828 autopsies (0.11 per cent.), whereas there were 10 cases from 139 autopsies on Chinese patients from that city. The higher incidence amongst Oriental peoples might account for the slightly higher figures from other American cities where there are large Chinese populations. For example, Wilbur, Wood and Willett (1944) reported 49 cases including 9 Chinese from 11,045 autopsies performed in San Francisco—an incidence of 0.44 per cent. On the other hand, the incidence among American Negroes was not significantly different from that in whites, which suggested that environment rather than racial origin was the important factor in the development of this disease in Africa (Kennaway, 1944). Bonne (1935), working in Batavia, showed that liver carcinoma was a common tumour of Malays and Chinese and Tull (1932) also demonstrated a high rate of occurrence among Singapore Chinese.

Morphology

Three groups, based on the gross appearance of 163 cases, were described by Eggel (1901). They were:

(a) solitary massive carcinoma—a single expanding and infiltrating mass, usually in the right lobe and accompanied by isolated secondary deposits elsewhere in the liver;

(b) multiple nodular carcinoma—the liver studded with numerous deposits of roughly equal dimensions;

(c) a diffuse carcinoma in cirrhosis indistinguishable, except by histological examination, from cirrhosis with hyperplasia.

Of these groups, multiple nodular carcinoma was the most frequent. Rolleston (1905), in his textbook of liver disease, incorporated a fourth group into this classification, a primary infiltrating or diffuse carcinoma, more diffuse than the solitary massive carcinoma described by Eggel.

The outstanding histological differences between hepatomata and cholangiomata were summarized by Hoyne and Kernohan (1947) and are illustrated in Table 1. Many writers have reported cases in which both patterns have been seen in the one tumour and, indeed, in single sections of it (Warvi, 1944; Lemmer, 1950). This demonstrates that the morphological form is the result of the direction of differentiation of cells and does not necessarily indicate their origin.

TABLE 1

Table showing the major histological differences between hepatomata and cholangiomata. (After Hoyne and Kernohan, 1947.)

	Hepatoma	*Cholangioma*
Arrangement of Cells	Cords and trabeculae	Tubules and alveoli
Type of cell	Large, polyhedral, with acidophilic granular cytoplasm	Small, cubical or cylindrical with clear basophilic cytoplasm
Stroma	Delicate capillary stroma	Massive fibrous connective tissue stroma
Bile secretion	Intracellular and extracellular	Extracellular

Metastasis

Almost every writer has commented on how frequently tumour thrombi are seen in branches of the portal and hepatic veins. Often tumour tissue has extended for considerable distances along main veins. Gregor (1939) found reports in the literature of 1 instances of tumour thrombosis of the inferior vena cava or right auricle, or both, i cases of primary liver carcinoma and adde one case of his own. Fox and Bartels (1928 reviewed 80 cases of hepatic carcinoma fro the English literature and found extrahepati venous extension in 22 cases—to the porta vein in 9, hepatic veins in 9, and to the inferior vena cava in 5 cases. Berman (1941) showed that light pressure on a carcinomatous liver would often express friable nodules which were, in fact, cancer thrombi from distended branches of the portal and hepatic veins. Histological evidence of venous invasion was frequently found and, even in parts distant from the main tumour mass, was usually gross. The percentage incidence of extrahepatic metastases seen in their cases was quoted by Eggel (1901) as 40 per cent.; Fox and Bartels (1928), 40 per cent.; Smith (1932), 60.9 per cent.; Berman (1941), 57 per cent.; Strong, Pitts and McPhee (1949), 42 per cent.; and Lemmer (1950), 50 per cent. The lungs and the portal lymph nodes were the most common sites of extrahepatic metastases.

A controversial subject has been intrahepatic metastasis and two schools of thought have arisen: one, that multiple nodules were intrahepatic metastases; and the other, that they were multifocal points of tumour origin. Muir (1908) and Yamagiwa (1911) concluded there was a progressive change from hyperplasia to neoplasia in multiple areas in a cirrhotic liver — the multicentric origin hypothesis. With liver-cell carcinoma occurring in haemochromatosis, Willis (1941) favoured the multifocal hypothesis, recalling multiple development of basal and squamous cell carcinomata in susceptible skins; there was a wide "field" of susceptibility to cancer which gave rise to multiple carcinomata simultaneously or in succession. Applying this to the liver he stated that the entire organ in haemochromatosis must be looked upon as potentially cancerous.

Ribbert (1909) presented the opposing view, that the multiple nodules were due to direct or venous extension within the liver from one primary tumour. Berman (1941)

observed many times a large tumour and small nodules immediately surrounding it and supported Ribbert's view. Winternitz (1916) stated that often tumour thrombi formed long strands in the intrahepatic branches of the portal vein and, at intervals, produced nodular enlargements. When this tissue was dissected from the veins little tumour remained in the specimen, thus supporting the intrahepatic metastasis hypothesis. Counsellor and McIndoe (1926) searched at the periphery of nodules for transition from normal liver to tumour cells and, when unsuccessful, concluded that primary carcinoma was unicentric in origin. Later they reversed their opinion when they discovered at one autopsy two nodules of similar type in each lobe of the liver, having shown, in earlier experiments, that the liver is a bilateral organ with no direct continuity between the vessels of right and left lobes (McIndoe and Counsellor, 1926).

Relation to cirrhosis

In 1942, Lisa, Solomon and Gordon recorded 6 examples of secondary carcinoma in cirrhotic livers from extrahepatic malignancies and found records of only another 5 cases in the literature. They emphasized the remarkable disparity in cirrhosis between the occurrence of primary carcinoma and of metastasis from extrahepatic malignancies.

A distinct causal relation between cirrhosis and primary carcinoma of the liver has been established. Berk and Lieber (1941), reviewing 1,073 cases of primary hepatic carcinoma, stated that the incidence of cirrhosis was 61.3 per cent. Stewart (1931) separated his cases into two groups and found the incidence of associated cirrhosis was 89 per cent. in hepatoma and 51 per cent in cholangioma. Berman (1941) found cirrhosis in every Bantu case studied, the great majority being liver-cell carcinomata. At first there was difference of opinion about the time relation between the cirrhosis and carcinoma. Hanot and Gilbert (1888) thought that both arose simultaneously in response to the same stimulus. Others, quoted by Ewing (1940), thought that the fibrous tissue was analogous with the stroma of a scirrhus carcinoma. Eggel (1901) and Rolleston (1905) put forward the modern opinion that cirrhosis is the primary change.

Stewart (1931) distinguished two types of fibrosis of the liver:

(a) post-necrotic scarring: replacement fibrosis after acute yellow atrophy with regeneration of the remaining liver cells to form large nodules. This process was eventually static;

(b) cirrhosis: toxic action long-continued with progressive fibrosis and hyperplasia of liver cells.

Stewart was not aware of a primary carcinoma arising in a liver, the site merely of post-necrotic scarring. Davies (1952) similarly divided his cases into two groups based on the type of hepatic fibrosis present but, apart from concluding that liver-cell carcinoma was clearly related to cirrhosis, no definite relation to the origin of the cirrhosis could be established. However, his evidence opposed the view that carcinoma developed only in rapidly regenerating livers and he thus agreed with Stewart (1931).

In haemochromatosis, too, this association of fibrosis and carcinoma has been seen. Stewart (1931) found 6 primary carcinomata in 52 cases of haemochromatosis collected from 5 British hospitals; these were included in Sheldon's (1935) monograph when he recorded a total of 26 examples among 363 acceptable cases of haemochromatosis — an incidence of 7.1 per cent. Wilbur *et alii* (1944) found 3 tumours in 20 cases in San Francisco, and Warren and Drake (1951) another 6 in a total of 20 patients with haemochromatosis.

TABLE 2

A table showing the sex incidence of cases of primary carcinoma of the liver.

	Males	*Females*	*Sex Unrecorded*	*Total*
Liver-cell carcinoma	27 cases	8 cases	1 case	36 cases
Haemochromatosis plus carcinoma	8 cases	0	0	8 cases
Bile-duct carcinoma	5 cases	9 cases	0	14 cases
Anaplastic carcinoma	1 case	1 case	0	2 cases
Total	41 cases	18 cases	1 case	60 cases

MATERIAL

The records of teaching hospitals of Melbourne from 1927 to 1954 inclusive were studied and 60 cases, for which there were adequate descriptions and available pathological material, were accepted from a total of 71. Five recent tumours were available for complete examination.

INCIDENCE

The distribution of patients according to sex and type of carcinoma is recorded in Table 2. To avoid confusion the terms hepatoma and cholangioma have been discarded and tumours composed of parenchymatous liver-cells and intrahepatic bile-duct epithelium are called liver-cell carcinoma and bile-duct carcinoma respectively. Liver-cell carcinoma occurred predominantly in males and, after excluding cases in association with haemochromatosis—a disease of males—the ratio of males to females was still significant, being 3.4 : 1. On the other hand, although the numbers were smaller, bile-duct carcinoma was clearly more common among females.

TABLE 3

A table showing the age distribution according to sex and the histology of the carcinomata. The range of age in each group is shown in parenthesis.

	Males	*Females*
Liver-cell carcinoma	63.3 yr.* (50–76)	60.5 yr. (42–84)
Haemochromatosis plus liver-cell carcinoma	63.9 yr. (58–74)	—
Bile-duct carcinoma	64.0 yr. (50–75)	59.2 yr. (27–79)

(* This average does not include one male infant of 9 months with a liver-cell carcinoma).

Five of the male patients with liver-cell carcinoma were Chinese and the remainder European in origin.

PATHOLOGICAL FEATURES

The tumours were divided, on gross appearance, into two distinct groups:

(a) nodular carcinoma;

(b) massive infiltrating carcinoma.

TABLE 4

A table illustrating the relation between cirrhosis of the liver and the gross morphology of the carcinomata, excluding the cases of haemochromatosis and carcinoma.

	Cirrhosis present	*No cirrhosis present*
Massive carcinoma	3 cases	17 cases
Nodular carcinoma	26 cases	5 cases
Other	0	1 case
Total	29 cases	23 cases

(a) The outstanding feature of the nodular group was the association with cirrhosis of the liver in no less than 26 of 29 cases (see Table 4). All gradations between a solitary, circumscribed, spherical mass on the one hand and multiple small nodules, indistinguishable from the hyperplastic nodules of cirrhosis, on the other were seen. In most specimens there were one or more large spherical nodules, usually in the right lobe, with smaller nodules of different sizes concentrated round it; but in others the nodules were about the same size and scattered throughout the liver. Some cases presented multiple confluent nodules, superficially resembling massive infiltrating carcinoma, but the nodules within the masses remained discrete. Generally the nodules ranged from 0.5 cm. to 10 cm. in diameter, were very soft and spongy and, on section, the central portions were often necrotic (Fig. I). In most organs, even though the patients were not jaundiced, the nodules were bile-stained; intact nodules were a light yellow-green and the necrotic areas a deep green or brown. In cases without cirrhosis the nodules were grey or pink in colour with patches of brownish discolouration from haemorrhage and/or necrosis. In 5 cases haemorrhage into a nodule was followed by rupture through Glisson's capsule and severe intraperitoneal haemorrhage. Blood-stained ascitic fluid was found in a further 5 patients. Tumour thrombi in the portal vein or its main branches were seen in 13 patients and in the hepatic veins in 3 of these (Fig. II). In some, a heavy concentration of tumour nodules was seen with a fan-like distribution

spreading from a point where the portal vein had been invaded and subsequently occluded by a tumour thrombus.

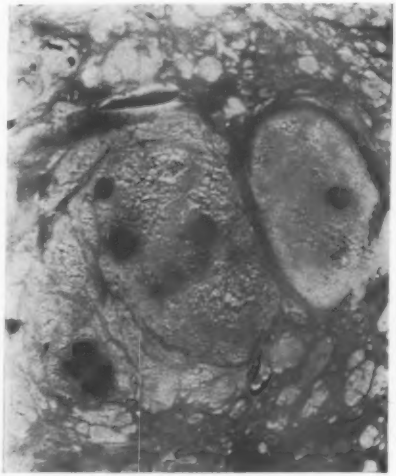

FIG. I. Photograph of a nodular liver-cell carcinoma in a cirrhotic liver showing haemorrhage and necrosis in the tumour masses.

(b) The massive infiltrating tumours were different. In each there was one large irregular mass of firm, grey, pink, or bile-stained tissue which sometimes replaced the whole of one lobe; haemorrhage and/or necrosis were infrequent. Often there were small spherical masses of similar tissue around the main mass resembling secondary deposits from extrahepatic tumours. Although sharply defined from the surrounding softer liver tissue, and in many cases multiple, these smaller masses differed from those of the nodular group by there being always at least one large infiltrating mass accompanying them and, in most, the tumour tissue was firm and light in colour. Furthermore, as shown in Table 4, the massive carcinomata were found most often in non-cirrhotic livers. Cirrhosis was present in only 3 of the 17 cases.

There were 8 cases of primary carcinoma among 35 patients who died with haemochromatosis—an incidence of 22.8 per cent. Six were multinodular, modified in form by the fibrosis peculiar to haemochromatosis. The connective tissue mesh in the liver in this disease is finer than in portal cirrhosis and, as a result, the islands of liver tissue are correspondingly smaller. Similarly the nodules of carcinoma were smaller than the tumour nodules found in portal cirrhosis.

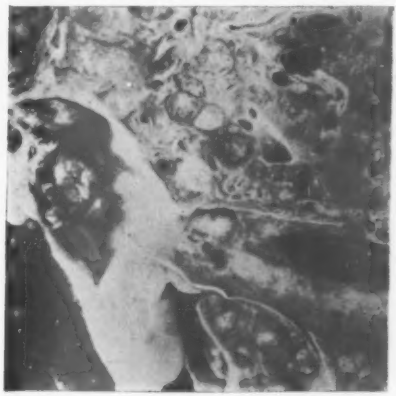

FIG. II. Photograph of tumour thrombi in the portal vein in a case of nodular liver-cell carcinoma complicating cirrhosis.

The carcinoma masses were firm and mottled pink in colour standing out against the dark red-brown background. The 2 remaining cases, in which the tumour tissue was of the same texture and colour, were massive carcinomata.

Finally one example, on gross examination, could not be included in the above groups. There was merely a uniform enlargement of the liver with slight increase in portal fibrous tissue. Subsequently this appearance was shown to result from a diffuse lymphatic infiltration of portal tracts by bile-duct carcinoma.

Histological appearances

Although the histological structure in any one tumour sometimes varied considerably, the majority showed one of two predominant histological patterns; the cells resembled normal liver cells (liver-cell carcinoma or hepatoma) or bile-duct epithelium (bile-duct carcinoma or cholangioma) respectively. Two cases were anaplastic tumours of uncertain nature.

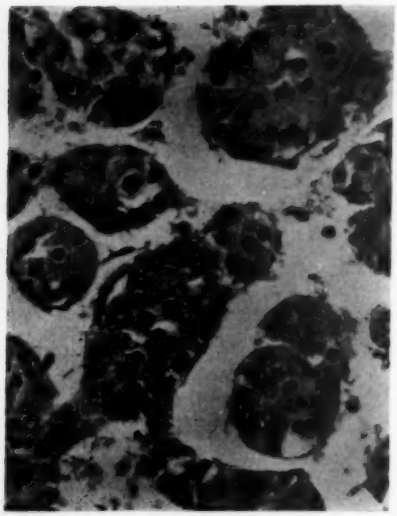

FIG. III. Photomicrograph of the pseudopapillary histological pattern showing small spherical masses of liver-cell carcinoma separated by wide sinusoidal blood vessels. (x 380)

(a) Liver-cell carcinoma was divided into two groups:

(1) Trabecular liver-cell carcinoma (31 cases). The characteristic feature of the majority, particularly those secondary to cirrhosis, was the resemblance of the cell arrangement to normal liver cords and of structure to normal liver cells. In place of connective tisue stroma seen in a carcinoma sinusoidal blood vessels were related to cell masses, just as normal hepatic sinusoids are related to the cells of a lobule. Although this resemblance to normal liver was usually well developed there were many departures from the typical trabecular pattern; however, in most specimens this arrangement was found in some part of the tumour. The liver-cell tumours could be arranged as a series, so that at one end of the scale there were those with a close resemblance to normal liver and at the other an anaplastic spindle-cell growth recognizable as a primary liver-cell carcinoma only because of some trabeculae in one area. Between the two one could observe a gradual diminution in the resemblance to liver cells or liver cords.

Within the nodules of highly differentiated tumours, cells were grouped in masses usually spherical or oval, as if suspended in a large cyst (Fig. III). However, they differed from the pattern of an intracystic papillary carcinoma in that the masses were roughly spherical whatever the plane of section; it was a pseudopapillary appearance. Furthermore cell masses were lined intimately at their periphery by a thin endothelium and the spaces between the cells were sinusoidal in type. The whole nodule appeared as a large cyst with cellular inclusions and the periphery of the "cyst" was lined by an incomplete multicellular layer of tumour cells. In 3 cases the cell masses were long convoluted trabeculae rather than spherical or oval groups (Fig. IV). This pseudopapillary trabecular structure was seen only in multinodular tumours in cirrhosis. In some parts of a cirrhotic liver every nodule was neoplastic whilst in others nodules of carcinoma and of hyperplasia were mixed haphazardly together. Though some tumour nodules were bounded by the general fibrous tissue mesh, others were separated by bands of atrophied liver tissue (Fig. V).

The cells of the pseudopapillary trabeculae were larger than normal and, in a few cases, were variable in size, shape and staining character and large tumour giant cells were common. The cytoplasm was usually granular and acidophilic; nuclei were larger than those of normal and hyperplastic hepatic cells. Sometimes there was little variation, indeed no more than in the cells of surrounding areas of hyperplasia. Moreover, the cells of some hyperplastc nodules adjacent to tumour areas showed variation in size, nuclear hypertrophy and hyperchromatism and often possessed more than one nucleus. Bile production was observed in many tumours. In 5 examples small alveoli resembling thyroid vesicles were found; they were distended with bile instead of colloid and lined by liver cells (Fig. VI). Stages of development of these alveoli could be observed, the earliest being the collection of minute amounts of bile between the tumour cells, in spaces analogous in position to normal bile canaliculi. As bile accumulated, these spaces gradually enlarged to form small cysts.

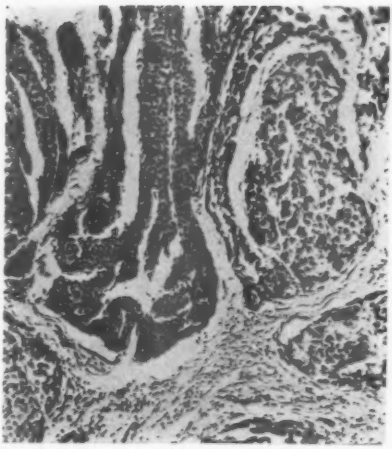

FIG. IV. Photomicrograph of a liver-cell carcinoma showing differentiation into long convoluted trabeculae. (x 90)

FIG. V. Photomicrograph of a trabecular liver-cell carcinoma showing a band of compressed and atrophied liver tissue between the mass of carcinoma and the fibrous tissue of the cirrhosis. (x 90)

In less differentiated tumours the cell masses were more closely packed and the diameter of sinusoids reduced. The histological picture of this step in loss of organization was also the next most frequent arrangement. Here nodules were composed of loosely packed, generally oblong cells in small groups of two or three (Fig. VII). There were but few structures resembling cords. Considerable variation in nuclear and cytoplasmic shape and staining and many mitotic figures were seen; many irregular tumour giant cells were apparent. Despite poor differentiation they were still recognizable as liver cells because of their large size, roughly rectangular outline, granular cytoplasm and rounded nuclei. Between the cells were numerous capillaries and sinusoids but no fibrous stroma. In some nodules, individual cells were independent, resembling a suspension in fluid. All the types of liver-cell tumour just described, despite their apparent diversity, were variants of a basic pattern characterized by differentiation towards trabecular formation, absence of fibrous stroma and a close resemblance of cells to those of normal liver.

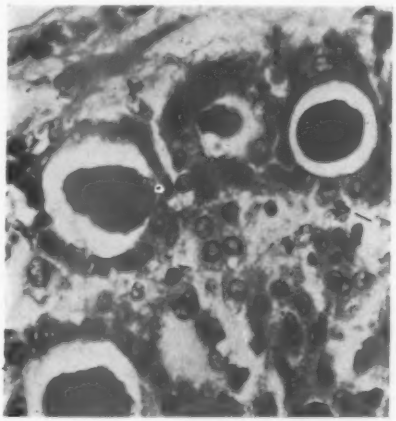

FIG. VI. Photomicrograph of a liver-cell carcinoma showing small alveoli of tumour cells containing collections of inspissated bile. (x 380)

(2) Spheroidal-cell carcinoma (5 cases). These cases of liver-cell carcinoma, each occurring in an otherwise normal liver, were significantly different from those already described—hence their segregation. Whereas in all previous cases there were some structures resembling liver cords, here there was none and the cells, in closely packed masses and sheets were separated not only by narrow vessels but also by connective tissue bands. Furthermore the cells were spheroidal rather than rectangular in shape and possessed prominent cell outlines. They were large,

some with granular and others with clear cytoplasm; all showed large vesicular nuclei. Many unusual forms of mitotic figures were seen (Figs. VIII and IX). These tumours followed closely the expected pattern of a carcinoma, possessing both cell masses and fibrous stroma. There was a definite relation between the histological pattern, the gross appearance and the presence of cirrhosis (Table 5).

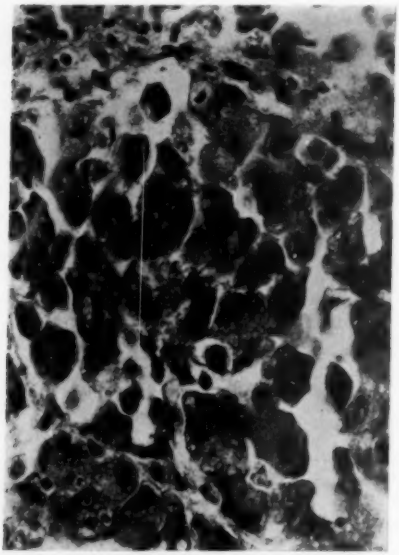

FIG. VII. Photomicrograph of a less differentiated trabecular liver-cell carcinoma showing large tumour cells with granular cytoplasm resembling normal liver-cells. (x 760)

TABLE 5

A table showing the relation between histological pattern and the gross appearance of liver-cell carcinomata and the presence of cirrhosis of the tumour-bearing liver.

Histological pattern	*Nodular*		*Massive*	
	Cirrhosis	*No cirrhosis*	*Cirrhosis*	*No cirrhosis*
Trabecular carcinoma	25 cases	2 cases	0	4 cases
Spheroidal cell carcinoma	0	1 case	0	4 cases
Total	25 cases	3 cases	0	8 cases

The trabecular pattern was confined almost entirely to nodular tumours of cirrhotic livers while the spheroidal cell pattern was a feature of massive infiltrating tumours occurring in previously normal livers.

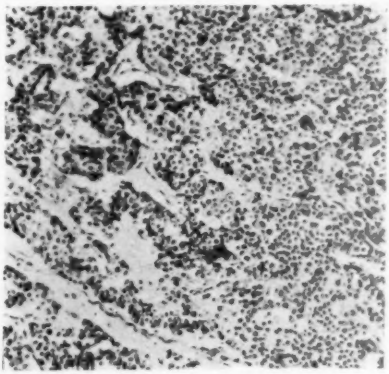

FIG. VIII. Photomicrograph of a more cellular section of a liver-cell carcinoma of the spheroidal cell type showing sheets of large spheroidal cells and many abnormal mitotic figures but with little attempt towards formation of trabeculae. (x 90)

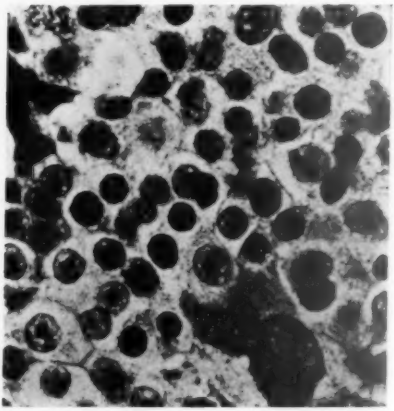

FIG. IX. A high-power photomicrograph of the tumour illustrated in Fig. VIII showing a section in which the cells more closely resembled normal liver cells. (x 760)

The tumours associated with haemochromatosis were all liver-cell carcinomata. Four of these presented a trabecular pattern but the remaining 4 cases had a disordered pattern not seen in other liver-cell growths. In these specimens the tumour cells were grossly irregular in size, shape, cell outline and in

nuclear form. The nodules were small and composed of loosely-spaced, almost stellate cells and syncytial masses of tumour giant cells (Fig. X). Haemosiderin granules were not found in the tumour cells.

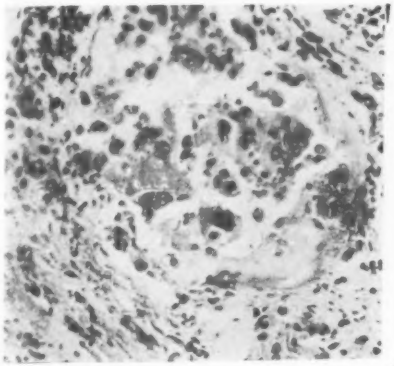

FIG. X. Photomicrograph of a liver-cell carcinoma complicating haemochromatosis showing the bizarre construction of tumour giant cells. (x 180)

In no case of liver-cell carcinoma was direct continuity observed between peripheral trabeculae of a tumour nodule and the cords of adjacent liver lobules.

(b) Intrahepatic bile-duct carcinoma. These tumours were predominantly adenocarcinoma and the histological appearances closely resembled those of extrahepatic bile-duct tumours, which have been reported elsewhere (Fleming, 1953). There were two distinct histological patterns:

(1) Columnar-cell adenocarcinoma (6 cases). These showed large irregular tubules and glandular spaces lined by tall columnar epithelium (Fig. XI). One case previously reported fully by Willis (1943) arose from congenital biliary cysts within the liver.

(2) Small duct carcinoma (8 cases). Here numerous small duct-like structures lined by low cuboidal or flattened epithelium occurred amongst disordered strands of spheroidal cells.

All these growths, like those of the extrahepatic bile-ducts, were scirrhus in nature. Generally the bile-duct-cell tumours were massive tumours unrelated to cirrhosis of the liver but, as shown in Table 6, not exclusively so. The table does not include the one case of small-duct carcinoma in which there was neither a nodular nor massive tumour but merely a diffuse hepatic enlargement.

(c) Anaplastic carcinoma (2 cases). These were small, clear-celled growths, spindle cell in places and of doubtful origin.

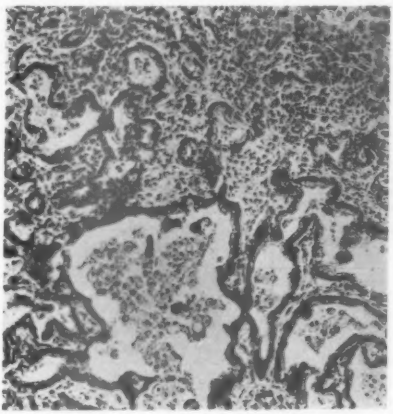

FIG. XI. Photomicrograph of a columnar cell adenocarcinoma of bile-duct type showing large acini lined by columnar epithelium set in a fibrous connective tissue stroma. (x 90)

TABLE 6

A table showing the relation of the histology of the bile-duct carcinomata with their gross appearance and the presence of cirrhosis.

Histological pattern	*Nodular*		*Massive*	
	Cirrhosis	*No cirrhosis*	*Cirrhosis*	*No cirrhosis*
Columnar cell adenocarcinoma	1 case	0	2 cases	3 cases
Small-duct carcinoma	0	2 cases	1 case	4 cases
Total	1 case	2 cases	3 cases	7 cases

Metastasis

Metastases to organs outside the liver occurred in 22 of the total of 60 cases, in 8 of 14 cases of bile-duct carcinomata, in 13 of 44 cases of liver-cell tumours (including those cases found at autopsy in patients with haemochromatosis) and in 1 of 2 anaplastic carcinomata. Bile-duct carcinoma metasta-

sized to lymph nodes, peritoneum, lungs, brain, bone and kidney and the metastatic behaviour followed the pattern expected from adenocarcinoma. In histological sections of the primary tumour, malignant cells were seen in lymphatics in 5 cases and in blood vessels in 5. The incidence of extrahepatic metastases from liver-cell carcinomata however, as shown in Table 7, demonstrated that the two groups previously identified possessed quite different metastatic propensities.

8 cases, to lymph nodes, peritoneum, lungs, pleura and bones, and histological sections showed venous involvement in 6 and lymphatic invasion in 3 of the primary tumours.

CLINICAL FINDINGS

The outstanding feature, in most patients even in those in whom carcinoma complicated cirrhosis, was the short duration of illness. Of the 25 patients with liver-cell

TABLE 7

A TABLE SHOWING THE INCIDENCE OF METASTASIS FROM LIVER-CELL CARCINOMATA EXCLUDING CASES IN ASSOCIATION WITH HAEMOCHROMATOSIS

Histology	*Nodular*				*Massive*			
	Cirrhosis		*No cirrhosis*		*Cirrhosis*		*No cirrhosis*	
	Metas.	*Nil*	*Metas.*	*Nil*	*Metas.*	*Nil*	*Metas.*	*Nil*
Trabecular carcinoma	3	22	1	1	0	0	3	1
Spheroidal cell carcinoma	0	0	0	1	0	0	4	0
Total	3	22	1	2	0	0	7	1

Extrahepatic metastases were found in only 3 of the 25 cases of nodular neoplasms with cirrhosis. Deposits in the lungs, and tumour thrombi in pulmonary vesels, were found in all 3 cases and, in one of them, there was spread to one suprarenal gland. This low incidence of extrahepatic metastases was the more remarkable because, in 19 of the total of 25 patients, numerous tumour deposits were found in the veins within the liver. In these cases intracellular bile accumulation was observed in metastases, particularly in the lung. There was no evidence of lymphatic spread in any of these specimens.

One liver-cell carcinoma occurring in haemochromatosis metastasized outside the liver to the ribs, spine, brain and pancreas and another spread directly along the falciform ligament. Malignant cells were found in the veins within the liver in 5 cases, but not in lymphatics. In this series (haemochromatosis) there was no demonstrable relation between incidence of metastasis and the gross appearance and/or histology of these tumours.

The second group, the massive tumours, behaved like the bile-duct carcinomata. Extrahepatic metastases were found in 7 of

carcinoma in cirrhosis, complete histories were available for 23 and the clinical presentations were as follows:

(a) Found accidentally at autopsy ..... 1 case
(b) Found at autopsy; death from haematemesis ..... 2 cases
(c) Long history of cirrhosis; rapid terminal illness ..... 2 cases
(d) Rapid progressive illness; no previous history 18 cases

The average length of illness, reasonably associated with the development of carcinoma, was four months and the longest illness was only one year. Typically, patients complained of progressive malaise, loss of weight, swelling of the abdomen, oedema of the ankles and pain in the epigastrium. Clinical jaundice developed as a terminal phenomenon but only in 3 cases. In the majority of cases, symptoms of cirrhosis prior to the final illness were so mild, if indeed present at all, that no comment was made about them.

The other types of primary tumour presented with similar short histories. The average duration of illness in bile-duct

carcinoma was five months, and of liver-cell growths without cirrhosis, three and a half months. In all but one case of carcinoma with haemochromatosis there was a rapid onset of a terminal illness with an average duration of six months. The remaining patient showed nothing more than an unrelenting progression of the symptoms of haemochromatosis.

DISCUSSION

It was apparent that the behaviour of certain types of primary hepatic carcinoma was unusual. Many authors have commented on the contrast between the biological features of liver-cell carcinoma and other cancers and some, cited by Ewing (1940), have doubted whether many cases included under this heading were indeed neoplastic. The features leading to this doubt have been well illustrated in this series. Among the liver-cell tumours there were two distinct groups of cases.

First, there was the group showing a multinodular appearance, cirrhosis of the liver, a trabecular histological pattern, heavy venous invasion within the liver and finally a relative absence of extrahepatic metastases. Secondly there was a group of massive infiltrating tumours of both the spheroidal cell and trabecular histological patterns, in non-cirrhotic livers, and in which there was both venous and lymphatic invasion within the liver, plus a high incidence of extrahepatic metastasis. This second group, like the bile-duct carcinoma, did not give major difficulties of classification because the pathological appearances and metastasis followed a more usual pattern. The group of nodular tumours with cirrhosis attracted special attention, for some specimens were at first difficult to identify as carcinomata.

There has been difficulty in identifying liver tumours because of uncertainty as to the true nature of some nodules; most authors have concluded that there is, in the cirrhotic liver, a gradation between nodular hyperplasia, adenoma and carcinoma (Muir, 1908; Winternitz, 1916; Counsellor and McIndoe, 1926; Ewing, 1940, and Willis, 1953). Winternitz (1916) observed that not only were adenomata and areas of nodular hyperplasia sometimes indistinguishable, but also, but for the presence of venous extension, adenomata and carcinomata might be histologically indistinguishable. In other words, the distinction between hyperplasia and carcinoma might depend on finding venous invasion or evidence of distant metastasis.

During this study, cases showing one or two small encapsuled hepatic tumours in livers without cirrhosis were observed in patients who died of various non-malignant diseases. These nodules were paler and firmer than the surrounding liver and on section showed, except for incomplete lobular formation, an almost normal trabecular pattern. There was no vascular extension and, in view of their nearly perfect differentiation, were reasonably called true adenomata. None of these tumours possessed the pseudopapillary structure. A search was then made of the main series to see whether there were equivalent benign tumours in cirrhotic livers, intermediate between nodular hyperplasia and carcinoma.

There was no question of the malignant nature of 19 of the 25 cases presumed to be liver-cell carcinoma complicating cirrhosis, since heavy venous invasion was seen in each. Attention was directed to the remaining 6 patients. Of these 5 showed multiple nodules, each possessing the characteristic tumour trabecular pattern. The last patient, known to have cirrhosis, died suddenly with gross ascites and had thrombosis of portal, splenic and mesenteric veins and of the inferior vena cava; but there was no evidence of tumour in the thrombus. One solitary nodule in this cirrhotic liver was larger and paler than the remainder and on section had a well-developed pseudopapillary pattern; but there was no evidence of venous extension. It was noteworthy that these liver-cell tumours complicating cirrhosis, whether venous invasion was present or not, possessed the same histological character—quite different from the pattern of nodular hyperplasia and also different from the adenomata seen in non-cirrhotic livers. It seemed logical to conclude that these tumours were examples of the same process and were indeed carcinomatous. Vascular extension might have been found in a greater proportion of cases had further sections been available. In no case was a nodule truly intermediate in histological structure, between nodular hyperplasia and carcinoma, found, although many times cells

of hyperplastic nodules adjoining frankly malignant nodules, or bands of hyperplastic liver around tumour nodules, did show nuclear hypertrophy and hyperchromatism. This phenomenon was called "collateral hyperplasia" by Ewing (1940).

Special histological or gross features were not seen in the 3 nodular metastasizing tumours complicating cirrhosis. A well-differentiated trabecular pattern similar to that in Fig. III was seen in two of these and yet, in other tumours not metastasizing outside the liver, there was poor differentiation and the cells appeared most "active." Most tumour thrombi in small veins, both within and outside the liver, were composed of small groups or sheets of polyhedral cells embedded in thrombus. This raised the suggestion that perhaps all nodules with the pseudopapillary and trabecular pattern were, after all, benign adenomata and that venous extensions and metastases arose from yet another less differentiated nodule, possessing the cytological features of malignancy. This possibility was eliminated when it was found that the trabecular pattern was reproduced in the extrahepatic metastases.

Intense venous invasion within the liver and yet so few metastases outside it was remarkable. If vascular extensions had been confined to branches of the portal vein the phenomenon could have been explained readily but, in many cases, hepatic vein radicals in continuity with the systemic veins were invaded. Ewing (1940) noted that loose intravascular tumour thrombi appeared early in tumours devoid of other malignant properties and suggested that, although the thrombi were capable of producing secondary deposits within the liver, they did not survive transfer elsewhere.

Doubtless some of the many hepatic nodules seen in these cases resulted from a fan-like distribution of tumour material peripheral to an invaded branch of a portal vein. Although intrahepatic metastasis might account for some tumour nodules, the possibility that many were individual carcinomata could not be excluded, especially when the nodules were of similar size and diffuse in distribution. It was the probable explanation when several masses were found to be confluent nodules; it appeared that each nodule represented a separate point of origin of carcinoma.

Why patients with cirrhosis should develop cancer of the liver is unknown, but work on this problem has been done in Africa where the disease is not rare. Most cases described there are liver-cell carcinoma complicating cirrhosis and the high incidence of cirrhosis at an early age has been held responsible for the remarkable frequency and age distribution of carcinoma among these people (Davies, 1952). Between 1925 and 1933 there were 253 cases of carcinoma recorded among Bantu labourers on the Witwatersrand gold fields and in Johannesburg, and of these no less than 229 (90.5 per cent.) were primary tumours of the liver (Berman, 1940). Berman (1941) also found multilobular cirrhosis in every Bantu case studied, listing the chief agents as helminthiasis, malaria, schistosomiasis, haemochromatosis and alcohol. Davies (1952) also noted the almost constant association of cirrhosis with liver carcinoma among young Africans but found that the mere presence of cirrhosis did not of itself explain the high frequency of neoplasia because, although the ratio of males to females with carcinoma was 6 : 1, there was no sex difference in the incidence of cirrhosis. There was some other contributory factor in males.

Impairment of hepatic function was very common among young Africans, male and female, although in not every case was there gross anatomical change in the liver. One result of impaired liver function was excessive oestrinization, frequently found in both sexes and almost constantly in Laennec's cirrhosis. An association between excessive oestrinization and the development of carcinoma was suggested since Schenken and Burns (1943) had induced "hepatomata" in mice by oestrogen administration (Davies, 1949, 1952). Davies (1952) pointed out, however, that a carcinogenic stimulus, whatever its nature, acted locally on the liver; there was no excessive incidence of carcinoma of other organs among native Africans.

In this present study definition of the type of hepatic cirrhosis was not attempted. In view of the changes in the classification of

cirrhosis over the years it would have been unwise and misleading to accept as valid today some of the stated diagnoses of early cases, particularly where descriptions of the morphology of the cirrhosis were brief or indeed absent. Nevertheless deductions concerning the general type were made from a study of clinical histories. In all but 2 cases the patients were in good health until the onset of a short terminal illness, about six months before death. It began, not with jaundice which might be expected to follow hepatic necrosis, but with slight malaise, anorexia, gradual swelling of the abdomen, loss of weight and mild epigastric pain—in other words, symptoms of malignancy. Jaundice appeared late and in only 3 of the 23 patients of whom an adequate history was available. The remaining above-mentioned 2 patients were known to have had cirrhosis for many years but they, too, succumbed to a short terminal illness during which jaundice was absent. Clearly, carcinoma of the liver was not a sequel of rapid regeneratve hyperplasia following acute or subacute hepatic necrosis. As Stewart (1931) suggested, carcinoma developed in patients with long-standing and progressive cirrhosis rather than in cases of post-necrotic regeneration.

It appeared reasonable that if patients with progressive cirrhosis did not die from liver failure nor from complications of portal hypertension all might succumb eventually from liver-cell carcinoma; in other words one might expect carcinoma to supervene on long-standing and progressive cirrhosis. The duration of the cirrhosis was unknown in most cases in this present study; most patients did not present until the onset of a rapid terminal illness. If the assumption that long-standing cirrhosis was to be found in elderly patients were correct, then carcinoma also would be found amongst older patients. In this series the average age at death of patients with carcinoma complicating cirrhosis was 63.4 years; over the same period, the average age at autopsy of a series of cirrhosis uncomplicated by carcinoma was 57.4 years.

There was even greater difference between the average age of patients dying of haemochromatosis (55.9 years) and of carcinoma complicating haemochromatosis (63.9 years), patients with carcinoma being almost a decade older. A similar finding was recorded by Warren and Drake (1951); the average age at death of their cases of uncomplicated haemochromatosis was 56.3 years compared with 65.5 years for those of carcinoma. They found no relation between the onset of carcinoma and the duration or severity of the disease but concluded that age was a determining factor in the development of carcinoma. It was noted that carcinoma of the liver in European communities occurred at an age when carcinomata of most other organs reached their peaks of incidence. Primary carcinoma of the liver may be yet another example of the operation of carcinogenic stimuli during this age period, in this case, on the cirrhotic liver. The occurrence of carcinoma at a much earlier age in African natives did not invalidate this argument. It was reported by Davies (1949) that carcinogens could be isolated from the livers of young Africans at this early age.

Davies (1952) dispensed with the usual classifications of primary carcinoma of the liver and stated that, from his experience among Africans, two types of carcinoma, nodular or massive, could be distinguished by naked eye. First, "hepatomata" were yellow-red rapidly growing tumours showing haemorrhage and necrosis in multiple, soft and spongy nodules, and secondly, "cholangiomata" were harder, grey-white or pink-white tumours in which haemorrhage and necrosis were uncommon. In the present series the nodular group of tumours were easily recognizable on gross appearance and it was possible to diagnose liver-cell carcinoma on those features alone, but liver-cell tumours occurred here in two forms. In the majority of the second group, which were massive infiltrating tumours, the tumour tissue was whitish and firm and the gross appearance resembled the description of bile-duct carcinoma given by Davies (1952). They were differentiated from bile-duct growths only by histological examination.

Their behaviour, too, resembled that of bile-duct tumours and so both varieties of tumour were considered together. It was concluded that most smaller spherical nodules surrounding the predominant infiltrating masses were true intrahepatic metastases and not multiple primary tumours; there was no reason to suspect a multicentric origin of

carcinoma when there was always one mass obviously much larger than the remainder. Extrahepatic metastases occurred in 4 of 5 cases of spheroidal type liver-cell carcinoma and from 8 of 14 bile-duct tumours. In both not only were intrahepatic veins invaded, but also lymphatics; there were secondary deposits in the regional lymph nodes, a feature not seen in this series in nodular liver-cell carcinoma of cirrhosis.

In each series, liver-cell carcinomata were more common than bile-duct tumours and were predominantly tumours of males. Everywhere the close relation between cirrhosis and liver-cell carcinoma was observed. Like carcinoma of the extrahepatic bile-ducts, tumours of the intrahepatic biliary system were more frequent in females. The slightly greater age of the Victorian cases was probably not significant. The outstanding

TABLE 8

A TABLE SHOWING A COMPARISON OF THE PRESENT SERIES WITH THREE AMERICAN AUTOPSY STUDIES, EXCLUDING CASES IN ASSOCIATION WITH HAEMOCHROMATOSIS

Author	*Liver-cell carcinomata*				*Bile-duct carcinomata*			
	Number of cases	*Average age*	*Cirrhosis present*	*Extrahepatic metastasis*	*Number of cases*	*Average age*	*Cirrhosis present*	*Extrahepatic metastasis*
Smith (1932) Chicago	17 (16 males)	57.4 yr.	9 cases	10 cases	6 (1 female)	54.3 yr.	0	4 cases
Loesch (1939) New York	12 (all males)	58.7 yr.	9 cases	7 cases	2 (0 females)	58.5 yr.	2 cases	1 case
Hoyne and Kernohan (1947) Mayo Clinic	20 (18 males)	58.7 yr.	15 cases	11 cases	11 (6 females)	56.9 yr.	2 cases	10 cases
Fleming (1955) Victoria	36 (27 males)	62.6 yr.	25 cases	11 cases	14 (9 females)	60.8 yr.	4 cases	8 cases

The final point to be clarified was whether the occurrence of the tumour in Victoria differed from that reported from communities of predominantly European origin elsewhere in the world. The conclusion reached was that there was no significant difference. During the period of study there were 13,826 autopsies performed at the Royal Melbourne Hospital from which 32 of the total of 60 cases of primary carcinoma of the liver were collected — an incidence of 0.23 per cent. Frequencies of the same order were found in other European communities by various authors, 0.11 per cent. in England (Stewart, 1931), 0.44 per cent. in San Francisco (Wilbur *et alii*, 1944) and 0.28 per cent. from Wisconsin (Lemmer, 1950). In general the morphology and behaviour of the cases presented in this study followed the descriptions of the writers reviewed earlier in this paper. A comparison of some details of this and certain American autopsy studies is illustrated in Table 8; the findings in each are similar.

difference was that the overall incidence of extrahepatic metastasis was much lower in the Victorian cases of primary liver carcinoma. Extrahepatic metastases were found in approximately two-thirds of the cases quoted by Smith (1932), Loesch (1939) and Hoyne and Kernohan (1947) from America, but in only one-third of the cases of this study. This was apparently due to the much lower incidence of metastasis from the group of liver-cell carcinoma in cirrhosis in the Victorian patients. In all series bile-duct tumours metastasized outside the liver more frequently than did liver-cell carcinoma. A similar comparison of cases of carcinoma complicating haemochromatosis showed little local variation from the findings in European communities reported by Stewart (1931) and Warren and Drake (1951).

CONCLUSION

It has been shown that, except for minor variations, primary carcinoma of the liver in Victoria conforms to the pattern of the

lisease established in European communities. Its behaviour amongst African natives, however, serves to emphasize its unique features and has established the group of liver-cell arcinomata complicating cirrhosis as something unusual in tumour behaviour. These inique features have been apparent in the present study, but the tumours have been so uncommon in this country that only after a period of more than a quarter of a century have sufficient cases become available to make such a study possible. The remarkable frequency of these tumours in young Africans, however, has provided material not only for statistical surveys but also for active cancer research; it is from this source that we are most likely to obtain elucidation of the problems which the study of primary carcinoma of the liver has raised.

SUMMARY

1. The current knowledge of primary carcinoma of the liver is reviewed.

2. Sixty autopsy cases from the State of Victoria are described, including 44 liver-cell carcinomata, 14 bile-duct carcinomata and 2 anaplastic tumours and their behaviour is compared with series from other parts of the world.

3. Attention is drawn to two distinct groups of liver-cell carcinoma:

   (a) a group of nodular tumours in cirrhotic livers possessing a trabecular histological pattern, in which extrahepatic metastasis is rare despite the almost constant and heavy intrahepatic venous invasion;

   (b) a group of massive infiltrating tumours in non-cirrhotic livers in which the metastatic behaviour is similar to that of other, more common, carcinomata.

4. Cirrhosis of the liver as a causal factor is discussed; it is concluded that carcinoma is a sequel of long-standing progressive cirrhosis rather than post-necrotic hyperplasia.

5. The remarkable frequency of liver-cell carcinoma at an early age in young Africans is related to the early and frequent occurrence of cirrhosis among them.

6. Eight cases of haemochromatosis complicated by carcinoma of the liver are discussed.

7. It is concluded that primary carcinoma of the liver in Victoria conforms to the pattern which the disease has demonstrated in European populations.

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REPAIR OF THE DEEP URETHRA

By Balcombe Quick

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FRACTURE of the true pelvis is complicated in from 5 to 10 per cent. of cases by rupture of the deep urethra. The injury may be to the membranous portion from laceration by bone ends or, at the prostato-membranous junction, either by similar direct trauma or by forcible dislocation backwards of the apex of the prostate from the deep layer of the triangular ligament. In this indirect form of injury the pubo-prostatic ligament is torn and the urethra severed by the resistant pelvic diaphragm.

In a lesion of this last type an accurate diagnosis is not always easy. Blood and urine are extravasated into the pelvis behind the pubes and an extraperitoneal rupture of the bladder may be simulated. Early and adequate investigation and treatment, moreover, may be difficult on account of associated injuries and shock.

With the continuing accumulation of blood and urine in the pelvis the whole bladder and prostate are forced further upwards and backwards and in this way there comes about a complete loss of alignment between the prostatic and membranous urethral segments. The torn ends may ultimately be found to be as much as 1½ inches apart.

The passage of a catheter into the bladder in such a case will have been found impracticable from the outset and an emergency cystostomy will have been carried out as an operative minimum. Thereafter any undue temporizing in the restoration of continuity of the urethra will exact heavy penalties in the organization of granulation tissue and the probable onset of infection. Increasingly great operative difficulties will then be experienced and even traction upon the bladder base and prostate by inflatable bag may fail to maintain approximation of the urethral segments. Thus the results are, commonly enough, poor although a temporary channel may be established. For not only does the mass of scar tissue encountered make accurate orientation difficult but it also renders the parts unresponsive to re-alignment. Finally a permanent cystostomy may come to be accepted as preferable to repeated operative failures. Winsbury White (1948) writes: "Reconstruction is an arduous task in cases improperly treated in the initial stages, when perhaps failure to restore continuity has led to retention of suprapubic drainage. In them will be found an indefinable fibrous mass occupying the interval between the rami and welded to them. It may extend to the bladder base and is often the seat of urinary fistulae. Ruthless removal of this mass is the first step, taking care to preserve any lumen that can be disclosed by supporting bougies, retrograde or passed from without."

The finding of an apparently unbridgeable gap in the urethra poses the final and crucial problem. Reference to urological authorities results in advice as to the fashioning of one or more flaps from the distal urethral segment to be swung to the apex of the prostate, attempting thus to supply a partial mucosal lining to the lumen or channel that is dissected out in the area of discontinuity. Those who have attempted to follow such advice may have regretted that their lack of skill prevented them from isolating a satisfactory and viable segment of urethra from the surrounding *compressor urethrae* or *corpus spongiosum*. Other writers have advocated the use of a segment of vein or portion of the prepuce as a free graft with, however, very doubtful success.

In the case under report a successful result followed the adoption of the well-known principle of the Esser inlay, and in view of the difficulties and disappointments attending other methods it appears to be deserving of further trial, for it seems that it may be relied upon to furnish the desired result of a fully epithelialized and permanent channel connecting the separated urethral ends.

The report of a single successful case, in advocacy of a method, is always of questionable value but fortunately similar happy results have attended two operations in which the same general principle was employed by

Rank and Wakefield (1950) and reported by them in the British Journal of Plastic Surgery.

The early care of the ruptured urethra will undoubtedly be undertaken by the general surgeon or urologist and since even the late and refractory case will usually remain in their hands it is to be regretted that their report has not had a wider circulation in a less specialized publication.

CASE REPORT

J.S., aged 19, was thrown from his horse, which rolled on him whilst face downwards on 25th August, 1944. He was operated upon that night at the Echuca Hospital with the diagnosis of fractured pelvis and an extra-peritoneal rupture of the bladder. He was admitted to the Alfred Hospital five weeks later, on 29th September, 1944, with a temperature of 100° Fahrenheit and pulse of 104. The accompanying report stated that when explored suprapubically "there was a great deal of extravasated blood about; a catheter that had been passed along the urethra was found sticking through a hole in the bladder posteriorly. It was extremely difficult to get close to the actual hole in the bladder but a tube was put in it but was not stitched in. The urethral catheter was left in and a tube placed in the *cavum Retzi*." (It is not clear if the bladder was also drained suprapubically at this first operation or some days later.) The catheter was expelled next day but for two or three days thereafter varying amounts of urine escaped *per urethram*. The report continued: "Unable to get a catheter into the bladder (early in September); still continues to pass small amounts of urine but has had a rocky time from sepsis, with increasing abdominal distension, controlled to some extent by sulpha-therapy."

On admission there was evidence of gross sepsis, both local and general, and during the first week after admission the temperature ranged up to 104.8° Fahrenheit. A blood culture proved negative. M. & B. 125 was given, 1 gram four hourly, and the urine alkalinized. A urethral catheter was passed and, although it did not enter the bladder, it was tied in and used to irrigate the paravesical area of infection. X-ray examination of the pelvis showed: "Old fractures of the superior ramus of the left pubic bone and through each ischio-pubic junction, uniting without marked displacement. The left hip joint space grossly narrowed — intra-pelvic dislocation of the head of the femur—arthritis, probably the former."

A week after admission, on 6th October, 1944, the report on a urethrogram was: "Urethra obstructed at membranous portion and posterior urethra not filled with lipiodol. Large irregular false passages pass obliquely from membranous urethra to bladder on left side."

An intravenous pyelogram on 17th October, 1944, showed little beyond the fact that lipiodol was still present in the left side of the pelvis. In the interim a sequestrum had been removed from this region, limited perineal drainage established and penicillin injections begun in the small dosage then in general use. The great improvement that soon followed with return of the temperature to normal, appeared to warrant some attempt at repair. Accordingly on 20th October, 1944, eight weeks from the date of accident, the old suprapubic incision was enlarged and the bladder opened more widely. Pus was seen to come up through the internal urethral orifice through which a catheter could not be passed for more than about two inches. By further perineal incision and dissection the site of rupture, just distal to the prostate, was found. It was then possible to pass the catheter through the prostatic urethra from the bladder to the perineal incision and thence along the distal urethra with the assistance of a sound passed from the meatus. The urethral gap proving quite unbridgeable, the use of an Esser inlay suggested itself. This rather hastily improvized operation was certainly ill-timed for reasons that then were not fully appreciated. The catheter upon which the graft was mounted remained in position until 22nd December, 1944, nearly two months, when it was withdrawn with considerable misgivings, suprapubic drainage being still maintained. Spigotting of this tube was followed by reasonably satisfactory micturition for eight days, after which failure gradually became evident.

A similar complete lack of success attended the next attempt to bridge the gap by means of pedicled grafts swung in from the margins of the perineal incision, this only resulting in a considerably prolonged stay in hospital.

It became realized that a probable common factor in the failure of both split skin and pedicled grafts to survive was insufficient enlargement of the fairway established through the scar tissue separating the ends of the severed urethra. As a result the contraction of this scar tissue, insufficiently excised or divided—the "ruthless removal" of Winsbury White—gradually resulted in a pressure necrosis of the graft tissue. The maintenance of this enlarged fairway calls for the use of a supporting tube for the Esser inlay of considerable diameter, greater than could be safely passed through the penile urethra, and just fitting the prostatic snugly. This tube, therefore, must pass from bladder to perineum only and not, as in the first operation, through the whole length of the urethra. The timing of this operation, also, was bad in that signs of infection were still present. In the presence of infection the application of split skin grafts is less likely to succeed, even with antibiotic cover, than a little later when granulations are just beginning to form.

With these considerations in mind operation was once more undertaken, on 4th April, 1945. Working both transvesically and through the bulbous urethra the track, again completely obliterated, was re-established by cutting down through the widely opened bulb upon the point of a sound passed through the internal meatus. The point of this sound could be located with reasonable accuracy by the tip of a finger passed up through the bulbous urethra. As it had not been possible to demonstrate the triangular ligament it was still uncertain whether the rupture had involved the membranous urethra itself or the

prostato-membranous junction. For this reason regard was paid to the probable direction of the fibres of the *compressor urethrae* when incising upon the sound, dissecting away scar tissue and enlarging the track by forceps dilatation. A tube which was of considerably greater diameter than the catheter used in the first attempt was then rail-roaded upon the sound from perineum to supra-pubic incision.

Twelve days later it was removed and a similar tube was dressed with a Thiersch graft over a length of about three inches of its middle portion. The graft was applied in spiral fashion, being secured to the supporting tube by a suture at each end. A light dusting with sulphanilamide powder was followed by freezing the graft with ethyl chloride spray. The grafted portion of the tube was at once drawn into the required position. Suitable steps were then taken to ensure immobility of the dressed tube, the single silkworm gut suture closing the perineal incision being made to assist in this important detail. Rather inadequate penicillin cover was again provided.

Fourteen days after operation the tube was removed and it was then found that a 12-15 sound passed easily and freely from bulb to bladder. A catheter was then passed from meatus to bulb and thence on into the bladder and tied in. By 12th May, 1945, the suprapubic incision was closed and dry and the perineal wound much smaller. A large sound passed easily on 17th May; but the catheter was re-inserted until its final removal on 25th May, 39 days after the inlay. Micturition was at once established normally although for a few days some drops of urine escaped involuntarily from time to time.

Following his discharge from hospital and subsequent removal of his home to another State all contact with J.S. was lost for over ten years and thus no assessment of the long term result was possible. Quite recently, however, he has reported for review. His bladder function, he states, is completely normal. The urine, passed in a full stream, is quite clear. There has been no episode of infection and his general health is excellent. A slight limp alone remains to remind him of his accident. He is married and the father of a three-year-old son, marital relations being quite normal.

The opportunity thus afforded for review is fortunate. It disposes, for one thing, of the fear that, as has been suggested, infertility necessarily follows such an injury and its repair. The result, in fact, is better than that which follows excision of stricture of the urethra, either post-gonorrhoeal or that due to trauma to the bulb, the "fall astride" rupture. The operation of excision, ribbonization, mobilization and suture at the hands of Hamilton Russell himself was, in fact, invariably followed by some degree of narrowing. A follow up of his own and other cases carried out many years ago showed that a passage of sounds at least annually was desirable and that in the interim the lumen would narrow down until a 6-9 sound would be very firmly grasped.

This case has been reported in some detail as illustrating the difficulties that follow any delay in early repair of the ruptured urethra. The result obtained by the use of the Esser inlay suggests its employment in the neglected case, and it is desired to emphasize that this method or operation calls neither for any particular aptitude in plastic technique nor any special surgical equipment. That designed and described by Rank and Wakefield, an ingenious plastic introducer acting both as a support for the graft and to prevent its disarrangement during placement may not be readily available to many surgeons. As a simple alternative the freezing method (Quick, 1943) of securing the graft during introduction is satisfactory and practical.

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THE SURGERY OF HYPERINSULINISM

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THE problems in the surgery of hyperinsulinism are twofold. First, it may be difficult to ensure that the condition is one of hyperinsulinism which may benefit by surgery. Secondly, it may be difficult to localize islet cell tumours or be satisfied that none is present.

This paper is the result of experience with four personal cases and four cases which have kindly been made available by colleagues.

Pathological Features

The usual lesion found in the pancreas in this condition is adenoma of the islet cells. This is found most commonly in the tail of the organ as a rounded, firm, plum-coloured lump, reasonably well demarcated, and about 2 to 3 cm. in diameter, although it may be larger (Fig. I).

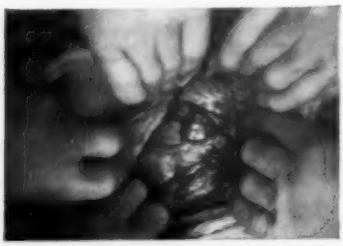

FIG. I. A photograph showing the macroscopic appearance of an islet cell adenoma (Case 6).

The histological structure is that of recognizable islet cells, with a dense nucleus and rather scanty eosinophilic cytoplasm (Fig. II). The cells may be arranged in groups (Fig. III) or, occasionally, in strands (Fig. IV); vascularity varies, but usually the tumour is fairly vascular.

Although the syndrome of hyperinsulinism is most frequently associated with benign adenoma, cases of an islet cell carcinoma with metastases and definite symptoms and signs of hyperinsulinism have been reported.

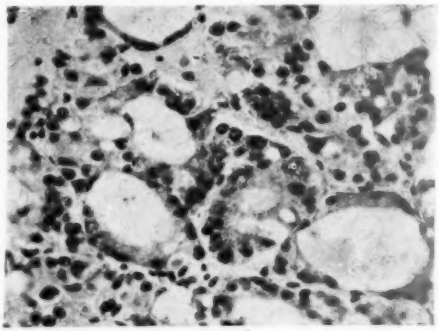

FIG. II. Photomicrograph of an islet cell adenoma, showing islet cells with dense nucleus, and eosinophylic cytoplasm (Case 6). (x 250)

The malignant tumour varies greatly in macroscopic appearance from the benign adenoma. The carcinoma is greyish-white, with areas of haemorrhage and softening, typical of carcinoma in general (Fig. V). Histologically, the main difference is in the cytological features — the carcinoma having pleomorphic islet cells and fairly numerous mitotic figures (Fig. VI).

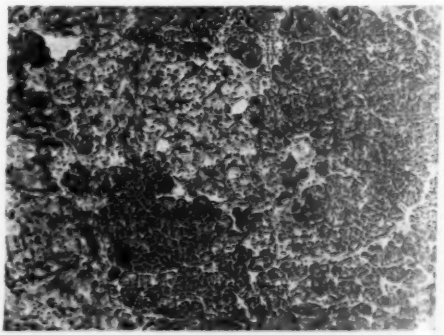

FIG. III. A photomicrograph of an islet cell adenoma showing cells arranged in groups (Case 6). (x 90)

On theoretical grounds, it could be postulated that an adenoma or carcinoma of the islet cells would cause the symptoms of excessive secretion of insulin. It might also be postulated that the same syndrome could be caused by hyperplasia of the islet cells without neoplasia. This has been found in practice. The syndrome was present but at operation no tumour of the pancreas could be palpated; but a portion of the gland was removed and histological examination revealed a surprising and unusual increase in islet cells in both number and size. The general structure of the gland was normal, but islands of Langerhans liberally studded each field (Fig. VII). The islet cells appeared hypertrophic and contained more cytoplasm than usual. Also, islet cells were found in some areas amongst the cells of the secretory alveoli in such intimate association that it seemed possible that the islet cell had actually arisen from the secretory epithelium.

This condition is apparently a true islet cell hyperplasia and, as far as could be judged from the sections, it was a generalized hyperplasia of well-differentiated cells, with the not unexpected effect of hyperinsulinism.

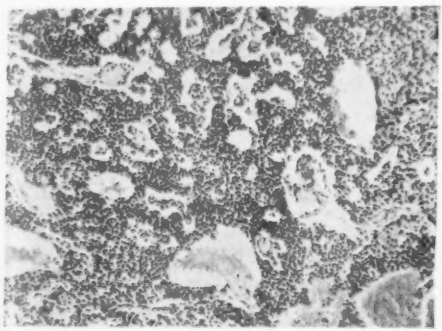

FIG. IV. Photomicrograph of an islet cell adenoma showing cells arranged in strands (Case 6). (x 60)

DIAGNOSIS

Hyperinsulinism is not common and, as a result, all cases had been under observation for some time before the correct diagnosis was made.

The patients complain of attacks of disorientation and, on many occasions, actual loss of consciousness. Their friends or relatives also describe air hunger, sweating and unusual behaviour during attacks. The attacks are followed by headache and a feeling of depression. The attacks are not considered by the patient to be connected with fasting and it is only on interrogation that a constant relation to fasting is detected. As a rule, the worst attacks are on rising in the morning, before breakfast or on exertion just after breakfast. In one patient, a trained nurse, the correlation with fasting was difficult to determine owing to the fact that her attacks occurred during night duty, when she ate little and worked hard. Very soon the attacks are found to be relieved or avoided by taking sweet food and it is not long before this routine produces embarrassing gain in weight.

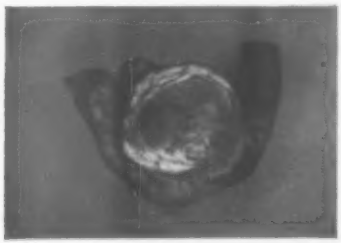

FIG. V. Photograph showing the macroscopic appearance of an islet cell carcinoma.

Changes in personality to a minor degree in between attacks are common and, coupled with the "funny turns," were the cause of most patients being referred to a neurologist or psychiatrist. The patients become very introspective and, with their relatives, are overjoyed when the possibility of surgical cure is discovered.

As a rule, there is nothing demonstrable on physical examination, but, in Case 1, the insuloma was palpable in the epigastrium; however, this is quite exceptional.

Investigations have been directed to satisfying "Whipple's Triad" (Whipple, 1944):

1. Attacks of nervous or gastro-intestinal disturbance which are induced by fasting.
2. The attacks are relieved by the administration of glucose.

3. The blood sugar level should be below 50 mgm. per cent. during an attack.

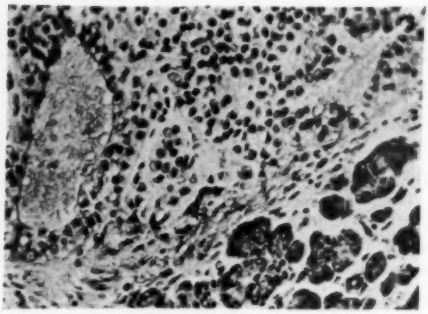

FIG. VI. Photomicrograph of an islet cell carcinoma showing pleomorphic cells, mitotic figures and intravascular penetration by tumour cells. (x 200)

To this triad should be added the characteristic prolonged glucose tolerance curve and abnormal insulin tolerance test (Conn, 1950). The fasting blood sugar is usually low, being in the vicinity of 50 mgm. per cent. The rise after the administration of glucose is not great being usually to about 100 mgm. per cent. The curve remains low and sinks below 50 mgm. per cent. between the third and fifth hours (Fig. VIII), and an attack will be induced if the fasting is continued. Sometimes a period of twenty-four hours of fasting is necessary to induce an attack.

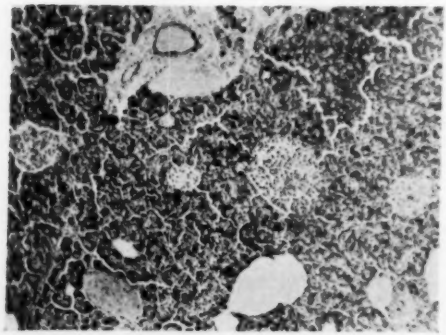

FIG. VII. Photomicrograph of islet cell hyperplasia showing the increased number of islands of Langerhans (Case 4). (x 60)

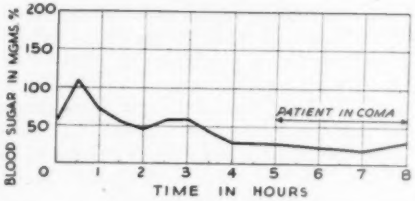

FIG. VIII. A prolonged blood sugar curve of Case 7 taken on 28th July, 1954, before operation and following the administration of 50 gm. of glucose.

These patients also have an unusual sensitivity to insulin. If a normal person is given an injection of 0.1 units per kilogram of body weight of insulin after fasting, the blood sugar level will sink to below 50 mgm., but will recover the normal level within two hours. In the patient with surgical hyperinsulinism, the recovery will not be complete after two hours (Fig. IX).

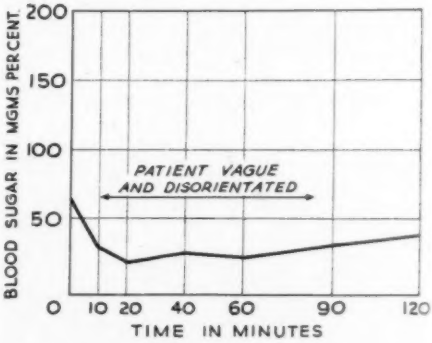

FIG. IX. A blood sugar curve following the intravenous injection of 0.1 units of insulin per kilogram of body weight in Case 7, carried out on 27th July, 1954, before operation.

TREATMENT

It is considered that, if "Whipple's Triad" is satisfied, the case can be regarded as one of surgical hyperinsulinism. Operation can be advised with confidence, and the result will be relief of symptoms. The problem remains, however, to determine at operation whether or not islet cell tumours are present.

The pancreas must be completely mobilized and examined by both sight and touch. If no islet cell tumour is discovered, then the correct procedure is to carry out a left hemi-pancreatectomy on the assumption that the

ondition is one of islet cell hyperplasia—he surgeon must be resolute in this decision. f one or more islet cell tumours are found, t is necessary to remove them widely, ncluding some normal pancreatic tissue ecause, owing to their irregular shape, it s difficult to enucleate the tumours. Another eason for removing them widely is the worrying feature of mitotic figures in the ells of many of these apparently innocent umours. However, the long-term follow up has indicated that the occurence of these nitotic figures does not effect longevity (Lopez-Kruger and Dockerty, 1947).

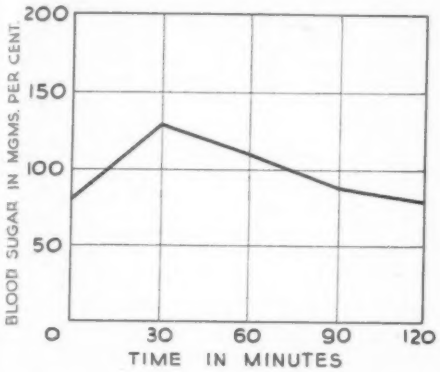

FIG. X. A blood sugar curve following the administration of 50 mgm. of glucose to Case 7 on 18th January, 1955, five months after operation.

In cases of islet cell hyperplasia, it is necessary to remove the left half of the pancreas. I have found that the task of preserving the spleen in these cases is quite difficult, owing to the frequent tearing of the splenic vein; so I have removed the spleen and splenic vessels towards the right, but preserving the inferior mesenteric vein. The pancreas is ligated and divided just to the left of the superior mesenteric vessels. The cut surface of the pancreas should be oversewn. I use "sixty" linen sutures for this suture.

Drainage is desirable. All cases drained a little pancreatic fluid for a few days following operation. In Case 7 there was considerable sloughing of the pancreas, and this resulted in a subphrenic abscess, which was successfully drained.

CASE REPORTS

Case 1

S.B., male, aged 55, presented on 15th July, 1947, complaining of attacks of dizziness and a feeling of impending disaster, usually occurring after breakfast when on his way to work. These attacks were relieved quickly by taking sweets. Physical examination did not reveal any abnormality. After 12 hours fasting, his blood sugar dropped to 50 mgm. per cent. although the patient did not suffer symptoms during this time.

In view of the hypoglycaemia, a laparotomy was carried out on 28th August, 1947, and a nodule was felt in the tail of the pancreas. The left half of the pancreas was removed. A microscopic examination revealed an islet cell hyperplasia.

The patient has remained symptomless since operation and, on 14th December, 1947, after an 18 hour fast, the lowest blood sugar reading was 90 mgm. per cent.

Case 2

E.W., a female, aged 60, presented on 18th August, 1947, complaining that for three years she had suffered attacks of sweating, weakness and inability to stand. The attacks were most common after breakfast, but also occurred after other meals. The attacks could be aborted by drinking very sweetened coffee. She had been treated as an epileptic.

Physical examination revealed a small mass palpable in the left hypochondrium. The fasting blood sugar was 40 mgm. per cent.

At operation on 25th August, 1949, the nodule was found to be a bluish tumour in the body of the pancreas and it was widely excised. Convalescence was uneventful. The symptoms have not recurred and the blood sugar has returned to normal levels.

The specimen was a firm, rounded mass, 1.5 cm. in diameter. Microscopic examination showed some resemblance to islet cell tissue; there was no definite capsule. The surrounding pancreatic tissue was compressed and occasional groups of tumour cells were seen in the connective tissue which appeared to separate the tumour from the main pancreas.

Case 3

R.O'C., female, aged 61, presented on 13th February, 1952, complaining of attacks of unconsciousness for 9 months. These attacks always occurred in the early morning about 6 a.m. A fasting period of 24 hours resulted in coma, with the blood sugar level sinking to 10 mgm. per cent. This was relieved by the administration of glucose.

On 16th June, 1952, a laparotomy was carried out and an islet celled adenoma was removed. The nodule consisted of cords of closely packed cells and alveoli of pale cells, with nuclei towards one end. In some places, definite lumens, even very tiny ones, were present but, in others, glandular structure could not be seen. Small rounded, calcified bodies were quite numerous. There appeared to be a connective tissue capsule around the tumour but some groups of tumour cells were seen beyond the main boundary line.

Her fasting blood sugar level, on 4th August, 1952, was 130 mgm. per cent. She made a complete recovery and has remained well.

Case 4

M.L., male, aged 69, presented on 7th July, 1952, complaining of attacks of unconsciousness for two years, occurring before breakfast, and occasionally at about 4 o'clock in the afternoon. He had noticed that his memory had deteriorated since the attacks began.

A physical examination revealed no abnormality. After a 24 hour fast, he passed into a coma, and his blood sugar, at this time, was 10 mgm. per cent. He recovered rapidly when glucose was administered.

As "Whipple's Triad" was present, a laparotomy was carried out on 23rd July, 1952. No tumour was found, so a partial pancreatectomy was carried out. A microscopic examination showed islet cell hyperplasia. Post operatively, on 1st August, 1952, his blood sugar after fasting was 90 mgm. per cent.

Case 5

V.L., female, aged 32, presented on 29th April, 1953, because she had fainted when on night duty. She had not eaten during the night owing to slight nausea. Physical examination at the time did not reveal any abnormality. She was kept under observation owing to continued anorexia and headache. Three days later she was found to be comatose, cold and sweating profusely. Her pupils failed to react to light and her eyes were roving. Apart from extensor plantar reflexes, her central nervous system appeared normal. The cause of the coma was in doubt but, as hyperinsulinism was suspected, she was given 20 mils of 50 per cent. glucose intravenously, with immediate recovery. A glucose tolerance test was carried out. The fasting blood sugar was 60 mgm. per cent., which rose to 100 mgm. per cent.; it then dropped to 30 mgm. per cent. after three hours, when the patient went into a coma, which was relieved immediately by 15 mils of 50 per cent. glucose given intravenously. The liver function tests were normal.

The patient then gave a history of frequent attacks of minor weakness and sweating, which were always relieved by sweet tea.

As "Whipple's Triad" was present, laparotomy was carried out on 24th June, 1953. No islet cell adenoma was discovered, and so a left hemipancreatectomy was carried out. The microscopic examination revealed an islet cell hyperplasia.

There was a temporary glycosuria post-operatively, with the blood sugar 180 mgm. per cent. She has remained well and has a normal glucose tolerance curve since then.

Case 6

M.B., female, aged 28, presented on 24th April, 1953, complaining of attacks of unconsciousness which had been observed by her neighbours and her husband. The husband noticed that she was twitching irregularly during the attacks.

She was observed in hospital next day, at 7 a.m. to be comatose and remained so until 11.30 a.m. when the result of a blood sugar was found to be 30 mgm. per cent. An intravenous injection of glucose resulted in the patient immediately becoming conscious. Physical examination was normal.

On 7th May, 1953, a 24 hour period of fasting caused loss of consciousness with twitchings. The blood sugar fell to 40 mgm. per cent. during this attack. There was rapid recovery when glucose was administered.

She was discharged on a high carbohydrate diet. She gained weight, but continued to have attacks of coma, particularly in the morning.

On 29th March, 1954, a laparotomy was performed, and a typical islet cell adenoma was discovered and removed (Fig. 1). She made an uneventful recovery. The post-operative fasting blood sugar level was 90 mgm. per cent. The tumour consisted of cords of closely packed cells. The nuclei were small and irregular, the cytoplasm of the cells was swollen and granular. In some areas there was variation in nuclear size and mitotic figures were present. No well-defined border or capsule was present, the tumour cells extended directly into the surrounding pancreas.

She has had no further attacks since operation and has completed a successful pregnancy. This is a satisfactory test of cure, as pregnancy causes hyperactivity of islet cell tissue.

Case 7

J. McK., male, aged 42, presented in 1951 with a history of "funny turns," which were considered at the time to be attacks of "petit mal."

Following the diagnosis, he continued to have attacks of depression and headaches. He found that glucose relieved the attacks. He began to have attacks of unconsciousness either just before or just after breakfast. His weight increased from 12 stone 7 pounds to 15 stone 2 pounds.

In July, 1954, he was investigated as a case of suspected hyperinsulinism. A prolonged fast, after 12 hours, induced a coma which was rapidly relieved by glucose. The fasting blood sugar was 50 mgm. per cent., it rose to 100 mgm. per cent. after glucose, and lowered to 50 mgm. per cent. at the second and third hours, and down to 19 mgm. per cent. after 6 hours. He was stuporose between the fifth and seventh hours. An insulin tolerance test showed that, after insulin, his blood sugar did not return to normal within two hours.

He was regarded as suffering from hyperinsulinism and a laparotomy was carried out on 10th August, 1954. No islet cell tumour was discovered and so a left hemipancreatectomy was carried out. There was some glycosuria during the immediate post-operative period.

His convalescence was complicated by a left subphrenic abscess, which was drained through the left thorax and was found to contain sloughs of pancreas. He has remained symptom free since, his

weight being 12 stone 5 pounds, and a glucose tolerance curve on 18th January, 1955, was normal (Fig. X).

Case 8

C.W., aged 17, presented on 16th November, 1954, complaining of attacks of unconsciousness followed by headache and depression almost every morning. His relatives stated that it was very difficult to rouse him in the mornings. He could be stood up but he could not support himself. He would recover suddenly but would complain of headache for hours afterwards. He had been treated by a psychiatrist as an epileptic, but possibly an hysteric. There was no abnormality detected on physical examination.

He was admitted to hospital on 20th November, 1954, for investigation. A prolonged fast induced an attack of stupor after 10 hours. His blood sugar at this time was 30 mgm. per cent. The attack rapidly disappeared after an intravenous injection of 20 mils. of 50 per cent. glucose.

A prolonged glucose tolerance curve showed a fasting blood sugar level of 60 mgm. per cent., which rose to 100 mgm. per cent., but fell to 30 mgm. per cent. after 3 hours and remained at that level until 6 hours, when the patient was semi-comatose, and an injection of glucose intravenously produced a rapid recovery.

At operation on 3rd February, 1955, a plum-coloured nodule was found in the body of the pancreas and was removed widely. No other abnormality of the pancreas was discovered. The patient made an uneventful recovery.

The nodule was a tumour composed of masses of cells, resembling islet cells arranged in a trabecular fashion. Groups of cells were separated by thick hyaline fibrous bands. There was no true capsule. The patient has had no further symptoms since operation.

SUMMARY

1. Eight cases of hyperinsulinism requiring surgery are described. All have made a complete recovery following operation.
2. The problems are: first, the diagnosis which depends on the establishment of "Whipple's Triad," and secondly, the demonstration of islet cell tumours. All patients in the present series fulfilled "Whipple's Triad" but, in four cases, the adenomata were found, and four cases presented islet cell hyperplasia.
3. Although mitotic figures are often seen in the cells of islet cell adenomata, this does not necessarily mean that the condition is malignant.
4. Treatment is wide excision of islet cell tumours or left hemipancreatectomy if no tumours are discovered.

ACKNOWLEDGEMENTS

I wish to gratefully acknowledge the information supplied to me by Mr. Julian Smith, Mr. John Turner, Mr. Douglas Leslie and Mr. S. F. Reid, who operated on four of these cases, and to Dr. Pincus Taft for the help given in the management of these patients. Dr. Whiteside of the Department of Pathology, University of Melbourne, and Dr. G. Harkness have given a great deal of time to the pathological studies of the specimens.

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TENOVAGINITIS STENOSANS AT THE CARPAL TUNNEL

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THE limited space in the carpal tunnel is shared by the median nerve and the common flexor group of tendons. Compression of the median nerve, with symptomatic or objective manifestations in the hand, results from encroachment upon the space from the boundaries. Where there is demonstrable local intrusion, for example, in arthritis of the carpus, the causative factor is obvious. In the majority of cases, however, the mechanism is not clear.

It is the intention of this paper to briefly review the literature and to examine the evidence presented by a personal series of twenty-eight cases for a mechanism to explain "spontaneous" compression of the median nerve in the carpal tunnel.

Review of Literature

Although the syndrome has been recognized as such only in very recent years, reports of cases presenting with various of its manifestations may be found in the literature of the late nineteenth century. Putnam (1880) reported thirty-one cases of nocturnal paraesthesia (acroparaesthesia) and noted that the symptoms in the hand were of median nerve distribution.

Hunt (1911) reported three cases, two bilateral and one unilateral, which showed considerable thenar wasting, of median nerve distribution, unassociated with sensory loss. He localized the lesion, theoretically, to the thenar motor branch of the median nerve at or under the distal extremity of the flexor retinaculum. Marie and Foix (1913) found bilateral thenar wasting in an elderly woman admitted to hospital following a cerebral vascular accident, which precluded examination of the hands for sensory loss. They dissected the wrists at autopsy and found swelling of the median nerve, proximal to the flexor retinaculum, with narrowing of the nerve at the point where it passed through the carpal tunnel.

The sex and age distribution of the bilateral condition was first pointed out by Dorndorf (1931) whose sixteen cases of thenar wasting and palsy were all women of climacteric age. He postulated that the pathogenesis was ischaemia, possibly in the median nerve, though he did not localize the site. He suggested that the selective effect upon the motor fibres was due to their greater vulnerability.

In a discussion on a case presenting with bilateral thenar wasting Moersch (1938) suggested that the thenar motor branch of the median nerve might be subject to trauma as it turns to the thenar muscles at the distal border of the annular ligament (flexor retinaculum). He noted, however, that paraesthesia and sensory changes were sometimes observed in these cases. He attributed these to a probable involvement of the main trunk of the median nerve at the annular ligament. Referring to atrophy, he speculated that relief might be obtained by "removal of irritating factors, or by surgical measures such as relieving pressure on the thenar branch of the median nerve, by section of the anterior annular ligament."

The motor phenomena were again noted by Wartenberg (1939) in seven cases of thenar wasting which he thought might be due to trauma to an anomalous thenar motor branch embedded in the distal fibres of the flexor retinaculum.

Both sensory and motor manifestations were present in two patients reported by Woltmann (1941). The first quoted was a woman with acromegaly in whom the symptoms and signs in the hand improved considerably after X-ray irradiation of the pituitary fossa. The second patient (a woman who was not acromegalic) was seen (in 1930) with similar clinical features. An X-ray of the wrist showed arthritis. Striking improvement in the symptoms occurred after division of the flexor retinaculum by Dr. J. R. (now Professor Sir James) Learmonth, and power eventually returned to normal. Before operation, a firm protuberance was observed over

the mid-ventral portion of the wrist, but no operative findings were given to indicate whether this was an enlarged median nerve or was due to arthritis. This operation, performed in 1930, is the first recorded example of decompression of the carpal tunnel by section of the flexor retinaculum.

Arthritis of the wrist following fracture was the culpable intruding factor in two cases of carpal tunnel syndrome reported by Zachary (1945). Both patients had objective median nerve changes in the hand. In the first the arthritis was due to old (bilateral) fractures of the carpal scaphoids, and in the second to a malunited Colles' fracture. Relief followed division of the flexor retinaculum in both cases.

In reporting thirty-eight cases Cannon and Love (1946) referred to the syndrome as "tardy median nerve palsy." All the patients in this series presented with varying degrees of thenar atrophy. Although sensory impairment was absent in ten, they observed that the onset was almost invariably characterized by paraesthesia; nine were subjected to operation. Four had suffered previous fractures of the wrist but in five cases no local factor was seen. One of the latter was acromegalic and another was the patient previously presented by Moersch (1938). In the nine patients explored, in addition to section of the flexor retinaculum, neurolysis of the swollen median nerve was also performed.

Brain, Wright and Wilkinson (1947) described six women (later increased to eleven in correspondence by Wright) all of climacteric age, exhibiting bilateral "spontaneous" median nerve lesions in the hand. They presented a comprehensive review of the literature (although they overlooked the contribution of Cannon and Love, 1946) and gave an excellent account of the differential diagnosis. The symptomatic relief obtained from section of the flexor retinaculum was striking, although motor recovery was slow.

Young (1950) reviewed 15 cases of bilateral acroparaesthesia, all of whom were women. With one exception they were all in the immediate post-menopausal age group. The exception, aged 71, dated her symptoms back over many years to this phase. Young remarked on the obvious association with the menopause and commented on the occurrence of the syndrome in pregnancy and acromegaly. He concludes that it is an endocrine disorder and he obtained consistent relief of the symptoms by the exhibition of oestrogens. He postulated that the mechanism of the syndrome was vascular and referred to the similar symptoms described by Weddell and Sinclair (1947) in arms rendered ischaemic by a sphygmomanometer cuff inflated to 150 millimetres of mercury.

Fourteen patients with median nerve palsy in the hand were described by Kendall (1950). He drew attention to the fact that in his series there were essentially two groups of cases. In those patients with unilateral palsy the duration of symptoms was short (average three months), the sex distribution equal, and the ages varied considerably. Adequate trauma, or other local factor, was frequently present in this group. In the bilateral cases the duration of symptoms averaged three and a half years, the sex predominance (5:1) was strikingly female and the age range only 40 to 53 years. Of those with unilateral palsy six of eight responded to immobilization of the wrist and protection of the palm from occupational trauma. The six bilateral cases were, however, severe and all required operative relief.

Of eleven cases reported by Phalen (1951) the syndrome was bilateral in nine, all of whom were women. Six patients in this series exhibited thenar atrophy, the others presenting with paraesthesia only. One patient also had De Quervain's disease and another stenosing tenovaginitis in the tendon sheath of the middle finger. Phalen comments on the similarity of these conditions and points out that they have the same sex distribution.

Kremer *et alii* (1953) first emphasized acroparaesthesia as a manifestation of median nerve compression in the carpal tunnel. They reported sixty cases presenting with acroparaesthesia, of whom forty were severe enough to warrant operation. Relief was obtained in thirty-seven, but in three, re-exploration was required. The findings in these are worthy of comment. In the first the nerve was markedly atrophic at both the initial and subsequent exploration. Slow improvement followed the second surgical attack but considerable sensory and motor impairment remained. Incomplete division of the distal portion of the flexor retinaculum, rectified at the second exploration, had caused

failure of improvement of the second case. In the third, a cyst arising from the flexor tendon sheath had caused re-occurrence of symptoms, and removal resulted in cure. The age of onset in this series was most commonly 40 to 50 years and the syndrome was five times more common in women than men.

CLINICAL MANIFESTATIONS

The most common and earliest presenting symptom is paraesthesia (acroparaesthesia). This term is used to describe the complaint of numbness, tingling, and painful burning in the fingers, often radiating up the forearm, sometimes even as far as the shoulder.

It occurs most frequently at night when the patient is in bed. In milder cases it is only present in the morning on waking and disappears rapidly. When severe, the patient may have to get up hourly in the night to wave the arm about to obtain shortlived relief. In some, in addition to the nocturnal manifestations, use of the arms during the day in knitting or sewing, sometimes in a trade, will exacerbate the symptoms. Posture of the shoulders or arms in bed at night seems to make little difference to the likelihood, or the severity, of the symptoms.

The paraesthetic area is frequently not localized to the median nerve distribution in the hand and the patients may state that it occurs in all fingers. Kremer *et alii* (1953) noted that many sufferers, if instructed to observe carefully the cutaneous distribution, would return to volunteer the statement that the little finger escapes. In association with the acroparaesthesia there is commonly complaint of stiffness and puffiness of the fingers. In some there is no objective change to be seen but others find that wedding rings become uncomfortably tight at night. The patient tends to drop things, and says that the hands are clumsy. This may be due to hypaesthesia, muscle weakness in the ball of the thumb, or stiffness of the fingers, possibly the result of restriction of movement of the flexor tendons in the narrow carpal tunnel (tenovaginitis stenosans) and, if this is so, it may be considerably aggravated by use during the day.

The signs present are quite variable. Wasting and weakness of the median nerve innervated muscles, that is, the opponens pollicis, and the abductor policis brevis, are common. Fibrillary twitching is absent. Anaesthesia, complete or partial, over the radial three and a half digits in the palm is variable, but can often be detected upon careful examination.

A lump or swelling over the mid-ventral portion of the wrist, proximal to the distal crease, is sometimes found. In some it is striking and, in one case reported below, there was also a visible and palpable swelling in the proximal hollow of the palm, resulting in confusion in diagnosis. Limitation of extension of the fingers, corresponding with the patient's complaint of stiffness, may occur. Tinel's sign is only occasionally found positive, but it might be expected to be present in patients exhibiting the detectable neuroma mentioned above.

The pneumatic tourniquet test described by Gilliat and Wilson (1953) has been found helpful as a confirmatory test in those cases in which the only manifestation is acroparaesthesia, and there are no objective findings. In this test a sphygmomanometer cuff above the elbow is inflated to above systolic arterial pressure and this produces or exacerbates in less than one minute, the paraesthesia in the median nerve territory. In the normal individual, similar but minor symptoms occur after two to three minutes; but these are felt over the whole hand, frequently first in the ulnar distribution but never present first in the median nerve.

PRESENT SERIES

Twenty-eight cases are presented in this report. These have been seen during the years 1949 to 1955.

Of these eighteen cases were treated conservatively and all these cases occurred bilaterally in women in the age range 37 to 53. Ten cases were subjected to division of the flexor retinaculum and these will be examined in detail later.

The eighteen conservatively treated cases all presented with acroparaesthesia only, without positive motor or sensory phenomena in the hands, and without any detectable swelling on the volar surface of the wrist. In the first seven patients seen, detail of the digital distribution was not sought. Seven patients were clearly able to exclude the little finger in their localization of symptoms in the hand, either

on first questioning or after a period of observation. Four were unable to localize the territory to the median distribution even after repeated observation.

In all these eighteen patients the symptoms were variable, having undergone remission or exacerbation over periods of up to two years, without apparent cause. All were treated by oral oestrogen therapy, in some supplemented by physiotherapy directed to the elevator muscles of the shoulders. The latter was used in the early years in the belief that, although the Adson manoeuvre was negative, some were costoclavicular in genesis. There was no extended follow-up in seven of the cases who were seen only once in consultation. Nevertheless they were all stated by their physician to have obtained symptomatic relief soon after the institution of therapy.

In the remaining eleven cases the results of treatment are known in some detail. In seven cases treated by oestrogens alone the response was good, insofar that most or all the symptoms disappeared on treatment extending over two to three months, with no relapse on discontinuing the treatment. In four, physiotherapy to the elevators of the shoulder was also given in addition, because of failure to respond adequately to the hormone. In two of these, symptomatic improvement occurred with no relapse on discontinuation. In the remaining two patients, relapse occurred on cessation of treatment, but remission was obtained with further institution of similar measures. It is probable that the last two, if seen in the later period of this review, would have been advised decompression of the carpal tunnel. Both were in the group in which the digital distribution of the symptoms was not clarified.

Only one of the conservatively treated cases available for follow-up was intolerant of stilboestrol given orally, and by changing the drug to dienoestrol the patient appeared to obtain improvement without the erratic bleeding engendered by the former drug.

The assessment of the results of the above conservative treatment is difficult because of the spontaneous variation in symptoms in this group, in which the manifestations were entirely subjective. Nevertheless the symptomatic improvement on treatment appeared quite convincing and the patients were grateful for the relief obtained.

Operation cases

The ten cases submitted to operation are now examined in some detail. In the first three of this group the symptoms were entirely those of bilateral paraesthesia (acroparaesthesia) sufficiently severe to interfere with sleep. There was no demonstrable sensory or motor change in the hand. All were housewives, with symptoms dating back six months, eight months and twelve months respectively. They were given oral stilboestrol and improvement on this regime occurred for the periods in which it was given—for periods of two months, three months and five months respectively. The decision to operate was taken because of intolerance of the drug in the doseage necessary for the relief of symptoms. In two, significant haemorrhage occurred and in the third fullness and oedema about the lower abdomen and hips. In one of the patients with uterine bleeding (in which treatment was managed by her own physician) it was apparently sufficiently severe to warrant hysterectomy. Whether the drug was necessarily responsible for this excessive bleeding cannot be stated as the patient was climacteric.

The fourth patient was a textile worker and dated her symptoms to shifting a case at work. She experienced a pain in the right wrist the same night to be followed by similar symptoms in the other hand three weeks later. When seen, she complained of puffiness and acroparaesthetic symptoms at night, together with increase in stiffness of her fingers when using her hands at work. This patient obtained no relief from physiotherapy and oestrogens.

In this group of four patients there was complete relief of all symptoms after division of the flexor retinaculum on both sides at the one operation.

Two further housewives aged 55 and 58, with symptoms extending over some months, had severe acroparaesthesia in the dominant (right) hand. The older patient, in addition, had stiffness in the fingers on use of the hand and all her troubles appeared during a period of increased household work necessitated by taking charge of a small grandchild. In both these patients plaster of Paris immobilization

of the wrist was used, in the one case as a temporary expedient for the relief of symptoms pending the patient's admission to hospital, and in the other because of her strong antipathy to operation. In each, freedom from symptoms continued for the period of immobilization. One patient had an X-ray of the wrist showing minimal lipping of the wrist joint, and in the other an X-ray was not taken. Both obtained complete relief following division of the flexor retinaculum.

Case 7

A medical practitioner, aged 38, had symptoms for several months. Acroparaesthesia, weakness, and pain of median nerve distribution were present on both sides on use of the hands. There was a previous history of rheumatoid arthritis, but there had been no activity in this condition for some years. The symptoms were disabling and bilateral division of the flexor retinaculum was performed with ultimate great improvement on the (worse) dominant side. In the other (left) hand, although symptomatic improvement was obtained, dryness, scaling and cracking of the fingers and hand present at the end of two years, indicated failure of recovery of the sympathetic component of the nerve. These persistent signs may have been due to incomplete division of the distal fibres of the retinaculum (which seemed complete at the time) or to scarring resulting from minor wound infection which unfortunately occurred on both sides but discharged through the wound with ultimate complete healing.

Case 8

Complained of severe paraesthesia in the dominant hand. This woman, aged 40, suffered from moderately severe dermatomyositis. She was, at the time of operation, being treated with Cortisone which was ameliorating her symptoms slightly. The stiffness of which she complained in the hand was probably due to the preceding disease as it was present on the other side, on which, however, she had minimal and variable paraesthesia. There was complete relief of symptoms from operation on the most affected side. Her physician advises that further symptoms on the second side have been controlled by local injections of hydrocortisone.

Case 9

Is open to some doubt because of the ultimate pathological findings. This man was aged 71 but was remarkably fit for his age and he was actively employed as a sweeper in a factory. He first presented with a history that, one week previously, while rolling a heavy drum, his right wrist became sore, and he had since suffered both nocturnal and diurnal pain over the median nerve distribution in the hand, worst in the thumb and middle finger. In addition he complained of stiffness and weakness in his fingers. On examination there was quite an extensive, diffuse, tender swelling in front of his wrist joint, with redness of the skin over the maximal point of swelling. He was unable to fully extend his fingers and on attempting to do so he experienced pain over the radial side of the palm of the hand. Tinel's sign was negative. There was detectable wasting of the ball of the thumb and partial anaesthesia of the radial three and a half digits in the palm. X-ray examination showed no abnormality apart from considerable calcification in the arteries. The wrist was immobilized in plaster and he was given antibiotic because of the redness and tenderness in the skin. On removal of the plaster fourteen days later the redness and local tenderness had disappeared, but the diffuse swelling remained. The plaster was re-applied for a further fourteen days and again removed. The paraesthesia had greatly improved but the stiffness in the fingers and the swelling of the wrist remained. The plaster was again reapplied for a further month at which time the swelling, though smaller, was still quite obvious. Paraesthesia had entirely disappeared. Median nerve compression was thought to be due to a deep tumour, proximal to the carpus, probably from tendon sheath. Exploration was advised. At operation through a longitudinal incision some three months after his first appearance, a considerable swelling of the median nerve was present, proximal to the flexor retinaculum. Deep to the nerve the tendon sheaths of the common flexor group contained a large amount of clear fluid. There was also considerable oedema of the sheaths themselves, the colour of which was the injected grey seen in tenovaginitis stenosans. Portion of the sheath was taken for section. The flexor retinaculum was then divided in the usual way. The pathologist reported that the tissue was granulation tissue and that it had the appearance of tuberculous granulation tissue. "There are numerous tubercles present of typical structure. Central caseation is not prominent. It is present in several tubercles, however, and these have the classical appearance of a tuberculous tubercle." In view of these findings the usual antituberculous and adjuvant treatment was instituted and continued for four months. For the first three months the wrist was immobilized in plaster. Over this period the lump disappeared entirely. The symptoms had disappeared on the immobilization of the wrist before operation. When last seen six months after operation the movement at the wrist and fingers was normal, there was no paraesthesia or anaesthesia, and appreciable recovery of the thenar muscles had occurred. He was considered fit for work. In the light of further experience the section was reviewed and although difference of opinion on the histopathology exists, it is probable that it cannot be regarded as clearly tuberculous. There is good clinical reason to believe that the findings would be consistent with *tenovaginitis stenosans* at the carpal tunnel with secondary effects upon the median nerve and the tendon sheaths.

Case 10

A girl, aged 23, complained (with some language difficulties) that she was suffering from pain in the right wrist and palm when working as a process worker in a printing establishment. It appeared to be confined to the median nerve distribution in the hand, and radiated up the forearm as far as the elbow. Stiffness in the fingers occurred after use of the hand, and the paraesthesia became worse. There was, in addition, nocturnal acroparaesthesia. On examination there was a large non-translucent semi-fluctuant swelling on the mid-ventral portion of the wrist extending for two inches proximally from the distal crease at the wrist. There was also a quite considerable enlargement distal to the flexor retinaculum in the proximal central concavity of the

palm. Crepitus was detectable at the wrist on flexing the fingers. There was quite marked wasting of the ball of the thumb and demonstrable anaesthesia of the palmar surface of the radial three and a half digits. There was appreciable limitation of extension of all the fingers, but not including the thumb. Tinel's sign was negative. X-ray of the wrist showed no abnormality. A median nerve lesion at the wrist was obvious and the diagnosis was confidently made of compound palmar ganglion although there was no evidence of involvement of the fifth finger extension of the ulnar bursa. The Mantoux test was, however, negative and X-ray of the chest showed no abnormality. At exploration a longitudinal incision was made proximal to the flexor retinaculum. The whole of the lump seen clinically was found to be gelatinous and swollen median nerve of quite astonishing proportions. It covered the common flexor group, extending to the flexor carpi ulnaris on the medial side, and radially, deep to the flexor carpi radialis as far as the radial artery. Dislocation of the nerve to examine the tendons showed the same oedematous tendon sheaths, containing fluid, as had been observed in case nine. A biopsy of the tendon sheath and the median nerve was performed, and the flexor retinaculum then divided. The patient made an uninterrupted recovery, with complete loss of symptoms and gradual improvement in thenar muscle power when last seen four months after operation. The pathologist reported oedematous nerve bundles in the portion removed from the nerve and chronic inflammatory oedema in fibrous tissue in the specimen removed from the tendon sheath.

DISCUSSION

A review of the literature indicates that a lesion of the median nerve in the carpal tunnel was first inculpated to explain the wasting and weakness of the relevant small muscles of the thenar eminence. Sensory loss was noted in some cases, but paraesthesia received only passing reference. Only in the last decade has this symptom achieved diagnostic significance. Only ten years ago Walshe (1945) concluded that nocturnal paraesthesia (acroparaesthesia) was due to abnormality at the thoracic outlet. One year later Cannon and Love (1946) observed that the initial symptom in their patients of "tardy median nerve palsy" was paraesthesia. In this series paraesthesia was elicited as a symptom in patients presenting with palsy. Kremer *et alii* (1953) reversed the emphasis by reporting a group in which acroparaesthesia was the presenting symptom and in whom, upon examination, a proportion presented with variable palsy or objective sensory loss.

In the earlier reported series reviewed, where compression of the median nerve at the carpal tunnel was recognized, a local radiologically demonstrable factor was incriminated (e.g., arthritis of the carpus). Reports of "spontaneous" compression appear with increasing frequency in the later years, and the striking sex linkage and age distribution in this group, frequently bilateral, has been the subject of repeated comment and speculation, regarding pathogenesis.

The six cases of bilateral spontaneous compression of the median nerve in the carpal tunnel reported by Brain, Wright and Wilkinson (1947) were all women of climacteric age. Young's (1950) fifteen cases of bilateral paraesthesia were all women in the same age group. A female sex predominance of 5 to 1 was reported by Kendall (1950) in bilateral cases. Phalen's (1951) series of eleven, included nine bilateral cases, all of whom were women. Kremer *et alii* (1953) noted a 5 to 1 female sex predominance with an age range of 40 to 50 years.

In the present series there were 26 women and two men. In 22 of the women, the condition was bilateral, and all were approximately of climacteric age. Of those with unilateral disease two older women (aged 55 and 58) had symptoms in the dominant hand. No significant local cause was seen in these two patients. Of the other two women, one had dermatomyositis with considerable thickening of the roof of the carpal tunnel (she also had minimal symptoms on the other side). The other had findings consistent with *tenovaginitis stenosans* at the carpal tunnel.

One man in this series who had previously suffered rheumatoid arthritis (but in whom X-rays were not taken) had bilateral manifestations. The other male, aged 71, showed findings — macroscopic and microscopic — which on review were not tuberculous but more probably *tenovaginitis stenosans* at the carpal tunnel.

In all the patients of the present series submitted to operation, enlargement of the median nerve was observed. In two it was detectable clinically and very large, and in others it was quite obvious at operation. This finding has been observed by many previous writers. Brain, Wright and Wilkinson (1947) point out that the swelling of the nerve is due to oedema, and this is confirmed by biopsy in one of the cases reported in this paper. They cite the similarity of the findings with those seen in tardy ulnar nerve palsy, proximal to the medial epicondylar groove.

There appears to be no doubt that an isolated lesion of the median nerve, manifest distal to the wrist, with motor palsy or weakness, or with objective sensory loss, is due to compression of the median nerve in the carpal tunnel.

There is now strong evidence that the symptom of acroparaesthesia, even in the absence of objective findings and after the exclusion of proximal pathology at the thoracic outlet or spine, is due to a similar mechanism.

Confirmation may be sought by the relief of symptoms obtained by plaster immobilization of the wrist, or by the pneumatic tourniquet test of Gilliat and Wilson (1953).

Pathogenesis

The carpal tunnel is a tendon compartment shared by the common flexor group of tendons and the flexor pollicis longus. These two have separate but usually communicating sheaths—the radial and ulna bursae. The tunnel also translates the median nerve from the forearm to the palm.

The boundaries of the tunnel are ligamentous. Posteriorly, the floor consists of ligaments covering the carpus. At each extremity the walls are ligament covered bones forming pillars, for the attachment of the stout ligamentous roof or flexor retinaculum, the volar surface of which gives passing attachment to the palmaris longus tendon (when present) and its extension into the palm—the palmar fascia. That there is relative narrowing at the carpal tunnel in this syndrome would appear undoubted from the evidence presented by — 1. The demonstrable intrusion upon the space from the carpus posteriorly in some cases; 2. The median nerve lesion distal to the tunnel; 3. The proximal oedematous "neuroma" of the median nerve; 4. The attenuation of the nerve seen at operation in the tunnel; 5. The occasional evidence, both macroscopic (at operation), and in function of the fingers, of the presence of stenosing tenovaginitis, involving the flexor tendon sheath; 6. The dramatic improvement following decompression by division of the flexor retinaculum.

The relative narrowing could be theoretically attributed to—a. Increase in volume of the contents; b. Decrease in volume of the compartment. There is no evidence in support of the first supposition.

Decrease in volume of the compartment may be from any or all of the boundaries: From the carpus floor the common factors are acute dislocation of the lunate or chronic arthritis following fractures of the carpus (e.g., scaphoid). From the lateral pillars local encroachment is rare and the only recorded example is chronic arthritis about the trapezium.

Intrusion from the volar boundary or flexor retinaculum might be possible. Thickening of the ligament was seen in one case (of dermatomyositis) in this series and one is reported by Michaelis (1950). In the personally observed case, however, there was a general disease in which thickening of connective tissues and collagenous tissues is extensively distributed, and might be expected to involve the ligaments of the walls and floor, no less than the roof, of the carpal tunnel. Similar deductions probably apply to the patient of Michaelis (1950) who suffered from rheumatoid arthritis. One of the two male patients in this present series, with bilateral manifestations of carpal tunnel syndrome, suffered also from rheumatoid arthritis.

In the majority of reported unilateral cases in males there was an occupational or local demonstrable cause to account for the encroachment upon the carpal tunnel. Where no local factor is demonstrable in males, and in unilateral manifestations in young and old women under similar circumstances, the suggestion is put forward that the essential mechanism is tenovaginitis stenosans in the tendon compartment, with generalized thickening of the walls of the compartment as in De Quervain's disease.

It remains to consider the mechanism of the bilateral "spontaneous" compression so commonly seen in women of the climacteric age, together with corroborative evidence in allied conditions. The consistent female sex distribution in the climacteric age has been emphasized previously. Endocrine factors have been postulated by several previous writers. The appearance of transitory symptoms of acroparaesthesia in pregnancy is common. Similar symptoms, often of minor degree, have a very high incidence in women between 40 to 55 years, many of whom do not

-eek treatment. More severe symptoms, fre-quently with signs as in this present reported -roup, bring the patients for relief. Acro-megalic patients with the syndrome are reported by several writers.

Thickening of connective tissue and liga-ments is shared by several syndromes men-tioned; these are — rheumatoid arthritis, dermatomyositis, acromegaly, pregnancy and the menopause. In the first three it is well known, and perhaps it is remarkable that there are no reports of the syndrome in myxoedema. When the softening of the liga-ments in pregnancy by fluid infiltration and the disturbances of fluid circulation in acro-megaly and at the climacteric are remem-bered, it comes to mind that an identical mechanism may be operative in these spon-taneous cases. Similar hormone mediated fluid distribution changes occur in many women, with premenstrual aching of the legs, increase in oedema of the arm following radical mastectomy, amongst others.

Support is given to this contention by the symptomatic improvement obtained by Young (1950) in his reported series, and by the response in many of the cases which form the basis of this paper with the exhibition of oestrogens. Further confirmation is obtained by the transient appearance of the syndrome, usually with spontaneous recovery, in climac-teric women, as the hormonal status is sorted out.

The writer has seen acroparaesthesia both precipitated and exacerbated in climacteric women in whom androgens were prescribed for secondary carcinoma of the breast and multiple spider varices.

From the evidence put forward the deduc-tion is made that, in the absence of demon-strable local factors causing compression of the carpal tunnel, the mechanism is general-ized narrowing of the tunnel by thickening of all the walls. This is a type of *tenovaginitis stenosans* (De Quervain's disease). The narrowing may be due to general diseases causing thickening of collagenous tissue (dermatomyositis) or to oedema of fibro-elastic tissue from hormonal factors in preg-nancy, acromegaly, and the menopause.

The reason for the increase of symptoms at night or the fact that they are confined to this period, has been the subject of several theories. Ischaemia of the nerve possibly from the arterio-sclerotic changes, is sug-gested by Brain, Wright and Wilkinson (1947).

The increase of ischaemic pain at night is well known in cases of ischaemia of the lower limbs due to vascular occlusion. These patients, like those under review, frequently adopt the practice of hanging their limb down outside the bed or walk about, to obtain relief of the nocturnal pain. From the similarity in the symptoms in these two groups of patients it would be reasonable to assume that the pain is of ischaemic origin and that the mechanism of the exacerbation at night is similar in the two syndromes. Nocturnal pain of ischaemic type is probably due to increase of metabolism when the limb is warm, which increases the relative ischaemia. It is usually quoted that the increase of meta-bolism in tissue is of the order of 7% for each one degree rise in temperature centi-grade. Alternatively the increased warmth may cause increased oedema and therefore increased pressure. It is probable that the first alternative is more likely.

The remarkable rapidity of the relief fol-lowing section of the flexor retinaculum in this disease is similar to that obtained, in nocturnal ischaemic pain in the foot, by any measure which increases peripheral blood flow.

The frequent complaint of stiffness of the fingers, with or without limitation of extension of the fingers and often increased by use of the wrist, makes the similarity between the present condition and that of *tenovaginitis stenosans*, remarked by Phalen (1951), quite noteworthy.

It is therefore suggested that spontaneous generalized encroachment upon the carpal tunnel by oedema or pathological change in the ligamentous wall, should be regarded as producing effects similar to those obtaining in *tenovaginitis stenosans*. The effects upon the median nerve, either symptomatic or objec-tive, are the unfortunate consequence of the inclusion of the nerve in a tendon tunnel.

TREATMENT

As a general principle, operative treatment should be advised in all cases exhibiting ob-jective signs of median nerve involvement in

the hand. These may be manifest as thenar wasting or palsy, or objective sensory loss in the hand. In severe cases the signs will be marked, and their demonstration will present no difficulty. In lesser degrees, particularly in bilateral cases, muscle weakness and wasting are by no means easy to assess, and sensation is often unimpaired even in the presence of quite marked wasting.

These factors may account for the large numbers of bilateral cases in which there appears to be no palsy, wasting, or sensory loss. In other words the identical lesion on both sides allows no norm for comparison and minor degrees of wasting and weakness are classified as normal. Nevertheless the patients with bilateral lesions show a high proportion of response to conservative treatment, and so the preliminary statement in this discussion on treatment should be interpreted in the light of these points.

Rarely there may be a detectable swelling over the mid-ventral portion of the wrist, and this finding, together with acroparaesthetic symptoms, is an indication for operation. Swelling of the nerve, detectable clinically, must be rare in the absence of demonstrable wasting, weakness or sensory loss.

Operation will be required in all females with unilateral disease and in all males, with unilateral or bilateral disease, if the symptoms are significant, even in the absence of objective phenomena. A general or local factor will usually be found.

In women with bilateral acroparaesthesia and no objective phenomena treatment may be conservative. Acroparaesthesia, usually minimal and transient, is common in pregnancy and at the menopause. The complaint may be no more than morning tingling, puffiness and stiffness of the fingers, which disappears soon after waking. When more severe, the patient may lose considerable sleep or state the stiffness, pain and clumsiness seriously interfere with work during the day.

Hormone therapy should be tried in this group. The drug used in this series was stilboestrol given orally. In pre-climacteric women the dose was 0.5 mgm. per day for the first half of the cycle, beginning after the last day of bleeding (usually for ten days). If the next was a normal period, and relief had not been obtained, the dose was doubled. This schema was continued, depending upon menstrual irregularity and relief of symptoms.

In post-climacteric women the drug was given continuously, and increased after an interval of ten to fourteen days much in the same manner as above, if symptomatic relief was not achieved. If the symptoms disappeared and the drug had been tolerated, trial discontinuation of the drug was made variably after three to six months. It was necessary to reinstitute treatment in some patients who relapsed following cessation of the treatment. In those patients who obtained no relief, or were unable to tolerate the drug or substitutes, or who relapsed consistently, operation was advised.

The wisdom of giving oestrogens to patients in this (climacteric) group may be open to some question. There is no doubt, however, that the indications in this syndrome are probably more decisive than those which determine the indiscriminate use of the hormone for many so-called "change of life" symptoms.

Operation

The operative procedure is very simple and can be performed in a few minutes. Both sides may be dealt with at the same time.

A sphygmomanometer is applied to the arm above the elbow, the limb is exsanguinated by an Esmarch bandage, and the cuff is then inflated to above systolic arterial blood pressure.

A transverse or longitudinal incision may be used. The former gives the better scar, but the latter gives the better exposure and was used where there was a large "neuroma" present. The transverse incision is one to one and a half inches long centred over the middle of the distal volar crease of the wrist. The longitudinal incision extends proximally for one to one and a half inches from the distal volar crease, on the mid-ventral line of the wrist. This incision heals surprisingly well and with no late disabilities. Some authorities (Bunnell) advocate continuing the incision into the palm and dividing the flexor retinaculum under vision, but this appears unnecessary and it places the scar on an unfortunate situation proximal to the hollow of the palm.

The palmar cutaneous branch of the median nerve which crosses the flexor retinaculum superficially, is in danger if the transverse skin incision is made too deeply at the first cut. The nerve should be identified in the interval just lateral to the palmaris longus when the latter is present. It is drawn to one side, usually the radial side. Palmaris longus is then retracted medially and the investing fascia of the forearm split longitudinally. The median nerve will be seen on division of the fascia, and it looks grey and odematous and often surprisingly large.

A grooved probe is then passed deep to the flexor retinaculum in front of the median nerve, as far towards the ulna side as is convenient. This is to avoid the origin from the flexor retinaculum of the short muscles of the thumb and to ensure that no damage is done to the thenar branch of the median nerve when dividing the distal fibres of the retinaculum. A tenotome is then slid down the probe, blade towards the retinaculum, and the ligament divided. After division a small artery forceps will now slide easily under the retinaculum and division of the distal fibres is ensured. The wound is then closed with skin sutures only and a pressure bandage applied for 24 hours. The patient may leave hospital next day.

Symptomatic relief is immediate and no disability results from the division of the retinaculum. Some patients have anaesthesia over the ball of the palm for a short variable time owing to traction on the palmar cutaneous branch of the median nerve. Several patients have commented on the improvement in the mobility of the fingers apparent after operation.

SUMMARY AND CONCLUSIONS

The carpal tunnel is a tendon sheath shared by a group of tendons and the median nerve.

Narrowing of the tunnel produces earliest manifestations in the median nerve. The first symptom is acroparaesthesia later followed by thenar wasting and weakness, and frequently detectable sensory loss.

Restriction of movement of the flexor group of tendons is commonly present.

Twenty-eight personally observed cases are examined. Evidence is presented that idiopathic or spontaneous median nerve compression at the carpal tunnel should be regarded as resulting from generalized circumferential narrowing of the tunnel, essentially similar to the changes in *tenovaginitis stenosans*. In this group the thickening of the walls may be due to general diseases causing swelling of the connective tissues, or to oedema the result of hormonal factors. In bilateral disease in women, where there are no objective phenomena, treatment may be by oestrogen therapy. If this fails, or the patient is intolerant of the drug, and in all others with significant symptoms or signs, operation should be advised.

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CARCINOMA AND RHABDOMYOSARCOMA IN THE SAME KIDNEY

A STUDY IN THE CLASSIFICATION AND HISTOGENESIS OF MIXED RENAL TUMOURS

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"Myself when young did eagerly frequent
Doctor and Saint, and heard great Argument."
Edward Fitzgerald, *The Rubaiyat of Omar Khayyam.*

IT is a long standing observation that tumours may histologically suggest a mixture of carcinoma and sarcoma. These growths have caused much dispute since the beginnings of histopathology, and still pose many problems which are not yet solved.

During the second half of the nineteenth century it was universally believed that carcinoma and sarcoma could co-exist in the same organ. Saltykow (1914) seems to have been the first to attempt a sytematic classification of such growths. He was followed by Meyer (1919) who quoted with approval Virchow's dictum that these double tumours could be derived from a stem cell "like two branches of one tree," and then outlined his own classification. Although now only of historical interest, both classifications deserve mention in some detail as their terminology, often unacknowledged, appears in many discussions of the subject. The classification of Saltykow named, amongst others: (1) "combination" tumours, sarcoma and carcinoma arising independently but mutually invasive, and (2) "mutation" tumours, the end results of sarcoma developing secondarily within the stroma of a carcinoma, or *vice versa*. Meyer's classification comprised: (a) "collision" tumours — Saltykow's first category, (b) "combination" tumours, made up of different portions derived from one stem cell (he instanced Wilm's tumour) and (c) "composition" tumours, in which both parenchyma and stroma had become blastomatous.

These historical hypotheses are now obsolete. The modern conception is that carcinoma cells are capable of existing in many forms, including spindle cells, and that many apparently "mixed" tumours are in fact carcinoma undergoing multiple differentiation. For the development of this idea no one person is primarily responsible. Its germ can be detected in earlier writings, for instance, Krompecher (1908); but its first strong assertion seems to have been by Ewing (1919), who instanced melanoma, basal cell carcinoma, adamantinoma, adenocarcinoma of thyroid or ovary, primary carcinoma of liver, and embryonal epithelial tumours as conditions where such spindle cell transformation could be frequently observed. According to his observations, favouring factors were rapid growth, relief of pressure, and inflammatory exudation. These general views were strongly supported by Kettle (1925) and Saphir and Vass (1938), who, after surveying 153 recorded carcinosarcomata, reached the following conclusions:

"In evaluating the seemingly sarcomatous features of the reported carcinosarcomas, the following complicating factors which play a role in the alteration of the fundamental histologic appearance of the tumours must be considered:

(i) variations of carcinoma cells, some of which assume spindle shapes and may be interpreted as cells of a spindle-cell sarcoma, a factor particularly true of 'squamous-cell carcinomas with transitional features';

(ii) marked anaplasia of the carcinoma cells;

(iii) chronic inflammation which (a) leads to morphologic changes of tumour cells, (b) produces much connective tissue which may be regarded as part of a malignant connective-tissue tumour, or (c) provokes a lymphocytic reaction sometimes taken as the lymphosarcoma component of some of these tumours."

It seems that the resemblance of spindle-cell carcinoma to sarcoma, which these authors discuss, does not stop at cellular morphology; it may also include the organization of intercellular substance. For instance, Willis (1948) is emphatic that no pattern of reticulin or collagen between cells is diagnostic of sarcoma, and the studies of Huggins (1931) and Foulds (1937, 1940) indicate a similar conclusion as regards cartilagenous and osteoid change. Using the rabbit as the experimental animal, Huggins found that urinary epithelium implanted into muscular tissue regularly caused the formation of bone. Foulds studied transplants of fowl carcinoma and found, in each successive host, the frequent development of cartilage or bone in close relation to healthy epithelial cells. In his own words: "The hyaline matrix corresponded with the pre-osseous substance described by Leriche and Policard (1926) and apparently it was formed by secretion or degeneration of the epithelial cells before connective-tissue cells entered it. There is little doubt that the connective-tissue cells, which later penetrated, formed bone rather than fibrous tissue as a result of their environment. It seems that the parenchyma cells of the tumour exerted the same individuating action on connective-tissue cells as the epithelial implants in Huggins' experiments and that osteogenesis was a dependent differentiation." These experiments can be supplemented by observations on the characteristic intercellular material of mixed salivary tumours (Masson and Peyron, 1914; Masson, 1922; Fry, 1927-28; Leroux and Leroux-Robert, 1934), all of which indicate that intercellular mucin, cartilage, or bone may occur in the stroma of a carcinoma and that the presence of such material in a "mixed" tumour does not in any sense indicate its origin.

It appears therefore that Saphir and Vass's (1938) main contention is reasonable and that almost all mixed tumours are more or less bizarre manifestations of multi-differentiating carcinoma. A residual group remains, however, for which this explanation is insufficient, and certain tumours of the kidney are a case in point. Here the problem is the occurrence of neoplastic epithelium combined with primitive muscle, as shown by the presence of longitudinal and transverse striations in spindle cells. To my knowledge, nobody has seriously suggested that an epithelial cell can develop these markings in their typical muscular form, though irregular concertina-like structures can occasionally be produced as an artefact. We are therefore justified in regarding such kidney tumours as true mixtures of carcinoma and sarcoma — one of the few undoubted examples of this combination which exists.

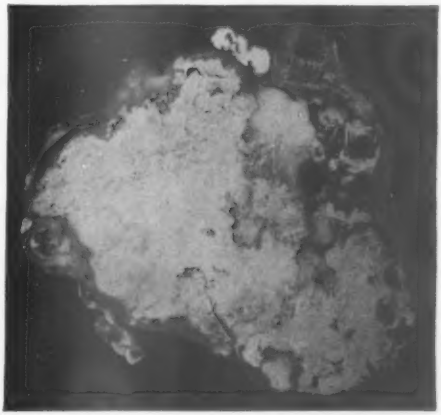

FIG. I. Naked eye view of double renal tumour.

In childhood, combined epithelial and muscle tumours of this kind are relatively common (Herzog, 1939). In the adult kidney they are rare, but authentic examples do exist. For instance, Hultquist (1938) reports carcinoma with leiomyosarcoma showing longitudinal fibrillae, while Hasner (1928) and Tedeschi (1930) have described carcinoma with rhabdomyosarcoma showing transverse striations. A tumour showing this second combination is recorded.

CASE REPORT

History: For 3 weeks before admission the patient, a woman of 54, had noticed a mobile mass in the left loin, accompanied by a persistent ache in the same area. Her previous history was not relevant and the only complaint of haematuria was that following an injury 8 years previously.

An intravenous pyelogram demonstrated prompt bilateral excretion of dye but a large soft tissue mass, partly calcified, was present in the origin of the lower pole of the left kidney; the upper calyx of the same kidney was correspondingly deformed and dilated. At operation (14th September, 1954) the left kidney was found largely replaced by a tumour adherent anteriorly to the descending colon. The mass was dissected out with difficulty. After the wound had healed, deep X-ray therapy was begun and continued for 5 months, eventually being stopped because of the poor general condition of the patient. Thereafter her course was steadily downhill, increasing signs of large bowel obstruction developing. Terminally, faecal and urinary incontinence appeared and she died on 12th August, 1955. Post-mortem examination was performed next day.

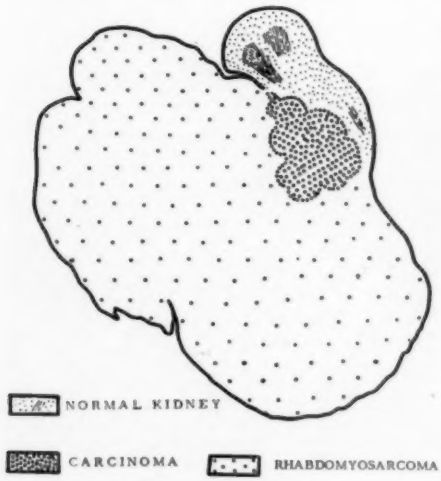

FIG. II. Key to Fig. I.

Biopsy specimen: This is a large mass weighing 960 gm. and measuring 14 inches in length. On section it shows a still recognizable upper portion of kidney and renal pelvis, with the proximal end of the ureter and a related lymph gland also identified. In the ureter is a small pinhead nodule.

The main tumour is composed of two parts. The portion projecting into the distorted pelvis is a papillary growth, one inch in diameter, which is only moderately vascular. Cross section of this shows a sharply delimited lobular structure, yellowish in colour with red areas of haemorrhage. Behind and above this lobular pelvic tumour the rest of the mass shows a grey, fibrillar periphery and a necrotic centre extensively infiltrated with chalky white deposits (Figs. I and II).

Post-mortem findings

The body is of an emaciated middle-aged female. In the left breast is a stony hard mass about one inch in diameter. The thyroid gland is normal.

Respiratory system: The trachea is dry and the right bronchus contains some mucopus. Both lungs are adherent posteriorly. There is an empyema containing 3-6 oz. of thin pus between the base of the right lung and the diaphragm. The right lung in relation to this shows a patchy bronchopneumonic consolidation; the left lower lobe is clear. In the upper lobe is a tumour mass approximately one inch in diameter which is adherent to the thoracic wall. The higher lymph nodes are not involved.

Heart and great vessels show no significant abnormality.

Liver and gall bladder: There is some nutmeg mottling of congestive type present in the liver. The gall bladder is normal.

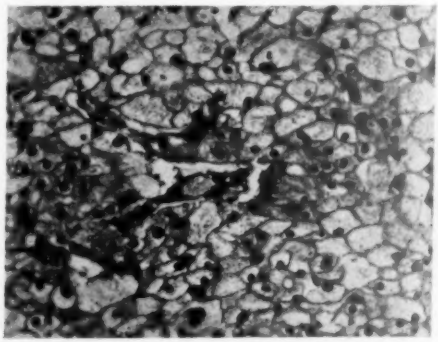

FIG. III. Detail of the carcinoma. (x 250)

Gastro-intestinal system: Oesophagus, stomach, small bowel are normal. The spleen, tail of pancreas, and hepatic flexure of colon are involved in a densely fibrotic mass, while a gangrenous cellulitis tracks retroperitoneally on both side of the abdominal aorta down to its iliac bifurcation. The para-aortic lymph nodes are not enlarged.

The spleen is of a pulpy consistence.

Right suprarenal, kidney, ureter, and bladder show no abnormality except that the mucosa of the renal pelvis and bladder is injected.

Uterus, left tube and ovary: The right tube and ovary cannot be found. The uterus is atrophic, with a calcified mass in its wall. The left ovary is also calcified.

Brain and meninges show no significant abnormal findings. Vertebral marrow is normal.

Histological examination

The biopsy specimen of the kidney shows a mass of papillary growth near the pelvis which is microscopically a primary renal carcinoma with inactive looking central nuclei, clear cytoplasm and well defined cell boundaries characteristic of its type (Fig. III); it is arranged in broad papillae. Corresponding with the naked eye appearances, the transition between it and the neighbouring growth is abrupt, the division being indicated by a band of collagen of varying thickness. The neighbouring sarcoma is a pleomorphic growth, varying greatly in the size of its cells and its nuclear morphology (Fig. IV) and demonstrating numerous mitotic figures. The cell boundaries are indistinct; the cytoplasm is arranged in wisps radiating from a central nucleus and seeming, in some cases, to branch and interlace with similar structures from neighbouring cells. There is extensive necrosis with numerous particles of amorphous calcification present not only in the dead material but also scattered between the living cells of both tumours. Although, as described above, there is a definite band of collagen separating the sarcoma from the carcinoma, in one or two places the collagen barrier disappears and the cells of the two tumours mingle. Where this occurs the tumours retain their separate identity, clumps of carcinoma cells being surrounded by sarcomatous elements (Fig. V). Similar appearances are figured by Hultquist (1938) in his reported mixture of renal carcinoma and leiomyosarcoma.

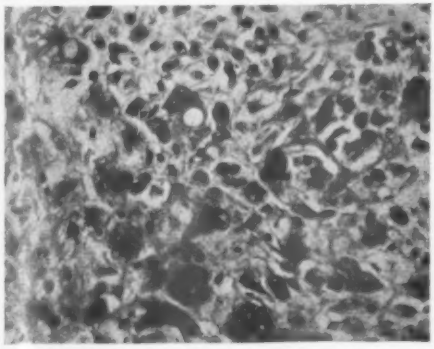

FIG. IV. Detail of the rhabdomyosarcoma. (x 250)

Special staining of the sarcoma cells by iron-haematoxylin (Heidenhain, 1896) frequently shows parallel cytoplasmic myofibrillae, each of which is regularly cross-hatched (Fig. VI). As these fibrillae twist in and out of different optical planes, they are not easy to see and consequently a cursory examination of any given cell only gives an impression of coarse granulation roughly disposed in a cross-striational pattern. The cross-hatched fibrillae may be sufficiently numerous to produce the effect of obvious striations spanning the breadth of the cell (Fig. VII), but such obvious striations are difficult to find, the markings usually being less complete. Of interest is the occasional suggestion of a "sheaf effect" in which a fine filament runs through the centre of the cell, giving off striations on either side (Wolbach, 1928) and also a "rosette effect" (Willis, 1948). It must be stressed that these striations are found in cells of grossly varied shape and size and that, in absence of demonstrable striations, such cells show no special morphological features. This is an interesting contrast to the findings of Stout (1946), Stobbe and Dargeon (1950), and Hurley (1954) in skeletal muscle rhabdomyosarcoma, where such special features are demonstrably present.

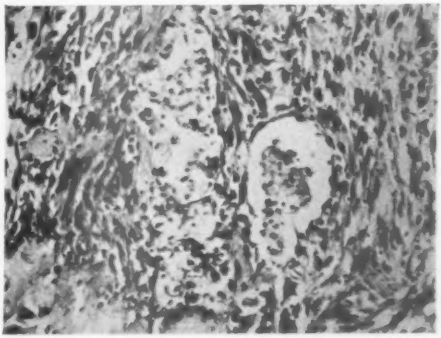

FIG. V. Clumps of carcinoma surrounded by sarcoma. Note how the separate identity of the tumours is sharply preserved. (x 120)

(Courtesy of J. V. Hurley.)

FIG. VI. Though not taken from the tumour described in the text, this cell illustrates how striations in general are constructed. Note the parallel cross hatched myofibrillae which in greater density would produce the appearance of striations spanning the whole breadth of the cell. (x 750)

The rest of the histological findings in the biopsy and post-mortem material may be briefly summarized. Recognizable sarcoma is not found in the immediate vicinity of the operation site but appears in retroperitoneal tissue surrounding the aortic bifurcation lower down. The nodule in the left lung is a secondary rhabdomyosarcoma. On the other hand, the ureteric nodule and neighbouring lymph node

are carcinomatous. The bone marrow is free of secondary tumour, the breast nodule is a calcified intracanalicular fibroadenoma, the uterine mass is a calcified fibroid and the ovary shows heterotopic bone.

In summary, this is a case of a double tumour of the adult kidney, being a mixture of papillary carcinoma and rhabdomyosarcoma. Secondaries from both tumours are present. The histogenesis and classification of this combined tumour has now to be considered and this, in turn, opens up the complex problem of mixed renal tumours in general, whether of adults or children.

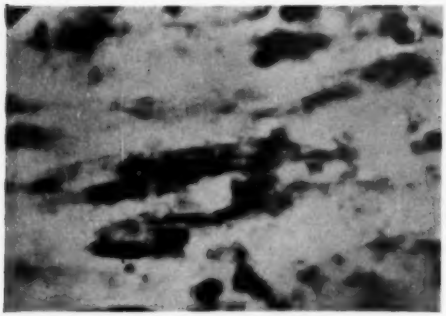

FIG. VII. Detail of rhabdomyosarcoma. A complex syncytial cell showing striations. (x 1800)

DISCUSSION

The juvenile group of "mixed" tumours is well known under the name of "Wilm's tumour" or "nephroblastoma," but other terms have been applied to them, such as "embryoma," "metanephroblastoma" (Potter, 1952), "mesoblastic nephroma" (Culp and Hartman, 1948), and a multitude of purely descriptive names summarized by these last two authors, together with many others in the German literature. As might be expected from the variety of nomenclature, our basic ideas about this group of tumours are in total confusion; the more we theorize, the worse the muddle grows.

The unfortunate fact about mixed renal tumours is that our theories so completely dominate us that we ignore even the demonstrable observations. Let anyone who doubts this consult a standard textbook on the subject of "Wilm's tumour." He is liable to find the tumour described in embryological terms which obviously prejudge the histogenetic issues. Spindle cells are described as "mesenchyme," cubical epithelium as "embryonic," acinar structures as "primitive tubules," "pro-glomeruli," and so on. These identifications may or may not be appropriate but what is so bad is their presence without qualification in what should be a purely histological description.

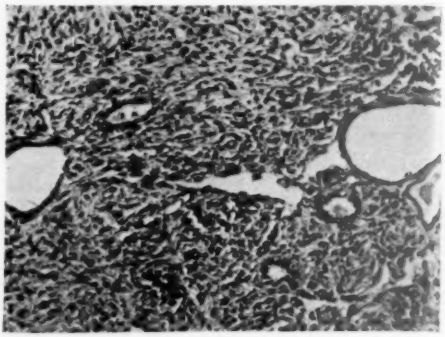

FIG. VIII. Detail of adenomyosarcoma. A group of acini surrounded by undifferentiated stroma. (x 120)

Stripped of such accretions, the histological picture of mixed renal tumours is found to be astonishingly simple. Take for instance those occurring in childhood, good descriptions of which can be found in the reviews of McCurdy (1934), Kretschmer and Hibbs (1931) and Herzog (1939). Apart from a small group of true renal teratomata (McCurdy), differentiation of these childhood malignancies follows a limited pattern. There is a groundwork of round or spindle-cell tissue which differentiates into masses of "epithelial" cells. These epithelial masses may be organized further into gland spaces, some indented or showing a pronounced tuft on one side. On rare occasions squamous epithelium (Muus, 1899) has been observed as also has a complex stromal relation with the epithelium of the pelvis and collecting tubules (Nicholson, 1931). However, epithelial differentiation may be poor, being represented only by a rosette formation in the stroma. This observation (Ribbert, 1836; Nicholson, 1931) has given rise to much highly imaginative speculation about "neurogenic" origins (Masson, 1938). Further differentiation of round or spindle-cell stroma may proceed in the direction of smooth muscle fibres; cross-striated elements are

common (Eberth, 1872; Herzog, 1939). Nerve fibres have been demonstrated by Masson (1938), but this is disputed by Willis. Fat, mucoid tissue, cartilage and bone (Hedrén, 1907) have also been observed, though more rarely.

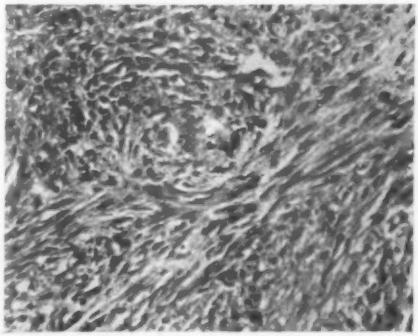

FIG. IX. Detail of adenomyosarcoma. Notice the differentiation of the stroma into distinctly visible bands of smooth muscle. (x 250)

A full duplication of the childhood picture is occasionally seen in adult life. A case recorded by Hasner (1928) showed striated muscle and acini of cubical epithelium, and Tedeschi (1930) reported a renal tumour composed of "hypernephroma," "angiosarcoma," cartilage and rhabdomyosarcoma. Hicks of the Royal Melbourne Hospital had a case, in 1949, of a renal tumour in a man aged 61 which demonstrates acini of cubical epithelium, a polymorphic stroma and smooth muscle (Figs. VIII and IX); in certain areas differentiation of the stroma into epithelial acini seems to be taking place (Fig. X) and the polymorphic cells of the stroma are occasionally transversely striated (Fig. XI).

Proved adenomyosarcoma of this kind is, however, rare in the adult. The more usual picture is of a double neoplasm, the one component of which is epithelial and the other a spindle cell of indifferent form which is characteristically pleomorphic. An appearance of merging or transition between the components is sometimes observed (Moore, 1951; Sissons, 1949; see also Fig X). Alternatively, one or the other component may appear as a focus of pure growth, and many varieties of this effect have been reported. Perhaps the most common is that the one component forms the main mass of the tumour, the other arising in small foci scattered throughout its substance. A typical case of this kind has been described by Weisel and Dockerty (1942). The present case, which presents two tumours almost completely separated by a collagen barrier, is an obviously related form, as also is the first case described by Chwalla (1936), where two apparently separate tumours were observed. These and other histological features of the adult group of these mixed neoplasms may be studied in the reviews by Hedrén (1907), Hultquist (1938), Esersky, Saffer, Panoff and Jacobi (1947), Culp and Hartman (1948) and Sissons (1949).

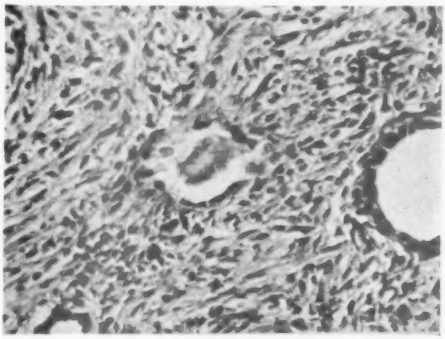

FIG. X. Detail of adenomyosarcoma. An acinus in process of formation from the stroma. (x 250)

How are these histological features to be interpreted? In the juvenile forms, a long line of writers, for example, Muus (1899), Meyer (1919), Nicholson (1931) and Delon 1935), have applied Virchow's original stem-cell hypothesis and postulated that the type cell of the tumour is multipotent, differentiating as it grows into epithelium or connective tissues such as cartilage, bone and muscle. This explanation accords so closely with the obvious histological features that it is widely accepted. Admittedly, there is still room for much argument about the nature and origin of the stem cell but the idea stated in general terms is reasonable and welds the juvenile group into a unity.

Mixed renal tumours of the adult may have a similar common stem-cell explanation but this concept is less easy to defend. The rare

complex malignancies cited above (Hasner, 1928; Tedeschi, 1930; Hicks, unpublished) are similar to the mixed tumours of childhood and, without undue strain on credulity, a common explanation may be accepted for them. However, certain other types of adult mixed tumour cause difficulty. For instance, the present case and the first one described by Chwalla (1936) might be considered as primary tumours arising separately and independently and coming into accidental contact. With this interpretation in mind, Culp and Hartman (1948) have deliberately excluded such "multiple" tumours from the general body of mixed renal neoplasms. Such an exclusion is surely far too rigid. Survey of the available cases indicates that this "multiple" appearance may be a separation and aggregation phenomenon, the histological "invasion" in the boundary zone (Fig. V) being due to incomplete separation of tumour components. Even if this explanation is not correct, and the tumours do arise separately, it is possible to regard them as multifocal rather than completely independent. That tumours can arise from multiple points of origin in a precancerous field has been emphasized by Willis; it is a matter of common observation in such conditions as basal cell carcinoma of skin and duct carcinoma of breast. Such a method of origin has actually been observed in juvenile mixed tumours of kidney, a case of Hedrén's (1907) being an outstanding example; so it need cause no surprise in the adult variety. I feel that whether the components of a mixed tumour are mingled in one focus or separate in multiple foci, they are still essentially the same tumour, subdivision on this ground being merely confusing. Culp and Hartman's (1948) opinions are therefore to be rejected and the common stem-cell hypothesis preserved.

A more serious objection to a unified concept of adult mixed renal tumours is advanced by Willis (1948) and Sissons (1949), who maintain that nearly all cases of "adult Wilm's tumour" are in fact multidifferentiating carcinoma. This may be true in some cases but its universal application is open to question. Observations on the present example and Hick's tumour indicate that any spindle or pleomorphic cell can be rhabdomyomatous and therefore such reported combinations as carcinoma with "spindle-cell sarcoma," giant-cell sarcoma," or "pleomorphic sarcoma" may possibly have been inadequately investigated adenomyosarcomata. Further to this the appearances which are interpreted by modern views as "multidifferentiating carcinoma" may in some cases be double differentiation from a stem cell. The general concept of unity therefore still remains and until some more solid basis of subdivision is brought forward there seems no adequate reason for introducing gratuitous complications.

FIG. XI. Detail of adenomyosarcoma. Stromal cell showing striations. Note the suggestion of Wolbach's "sheaf effect." (x 3300)

Granted that all mixed renal tumours are equally the result of stem-cell activity, it remains to enquire into the nature of the stem cell and its origin. Here the comments of Foulds (1940) deserve full quotation: "Tumours perhaps originate, on occasion, at every stage of development, but embryology has been too often invoked in the interpretation of complex tumours. As a rule, the plausibility of embryological theories is inversely proportional to the measure of the information available about the capacities of normal adult cells." The application of this general statement to the mixed renal tumours is obvious. For fifty years it has been assumed that these growths are derived from embryonic tissue. Is this in fact true?

The assertion of an embryonic origin for any given tumour is usually based on a characteristic age incidence and histological picture. The observations on the age incidence of mixed kidney tumours are well known. They can arise in the foetus (Wells, 1940) but are typically a condition of early

childhood, being most common before the age of 3. Few appear after the age of 10 and adult examples are rare (Ewing, 1919). It is generally held that an origin from persistent embryonic blastema could explain this age distribution.

This may be so, but closer inspection reveals certain disturbing features. Take for instance the occurrence of mixed renal tumours before birth. This is sometimes mentioned as evidence for an embryonic origin; but what is not correspondingly stressed is the extreme rarity of these neoplasms. Potter (1952), in an experience of 19,000 autopsies on children dying under one year of age, has not found one example, and Wells (1940) was only able to discover 5 in the whole of the world's literature. Potter considers this scarcity of cases as "somewhat surprising." On the basis of currently held embryonic theories, it is certainly very difficult to explain.

Again, embryonic tissues are held to persist into childhood, so causing the tumours at their peak incidence. Unfortunately, this hypothesis is often put forward with scant reference to the normal development of the kidney over the period in question. It is true that subcapsular embryonic mesenchyme may persist after birth, and a good deal of information is available about this structure —the "metanephric cap" or "nephrogenic zone." The formation of glomeruli within this zone forms a striking histological picture (Fig. XII). The point at issue is how long the metanephric cap can persist beyond foetal life, and this has caused some debate. Maximow and Bloom (1948) consider that it disappears six to eight days after birth. Felix (Keibel and Mall, 1912) extends the period to ten days, while Jordan and Kindred (1942) quote Toldt and Hauch's opinion that this structure may persist for three months. Potter and Thierstein (1943), after studying 1,000 infants and foetuses, reach a general conclusion that the disappearance of the metanephric cap is strictly related to physical measurements such as weight and length and is only secondarily influenced by age. Formation of glomeruli, in their experience, almost invariably ceases when the infant's weight is between 2,100 gm. and 2,500 gm., that is to say, somewhat below the normal 3,000 gm. birthweight. Consequently the fully mature, well-nourished baby delivered at full term does not possess a metanephric cap, and its presence is an excellent pointer to immaturity. The longest period after birth at which the cap has been observed is 69 days, but such a lengthy period is exceptional. According to Potter (1952), after the disappearance of the subcapsular blastema the kidney grows by lengthening of tubules and increase of interstitial tissue. We therefore conclude that at the time of maximum incidence of mixed renal tumour no embryonic tissue is normally present in the kidney, growth occurring by the proliferation of adult cells.

Of course it is possible that blastema may exceptionally persist, or that a tumour derived from blastema in the neonatal period may not become apparent till much later, but both these arguments reek of special pleading and are very inadequate to explain mixed renal tumour in older age groups, especially adults. To meet this admitted difficulty Potter (1952) proposes that the persisting tissue is not normal subcapsular blastema, but abnormal mesenchymal "rests" deep in the substance of the kidney. She has claimed to find such structures in two cases, one a very premature infant who died at birth, and the other a still-born foetus with polycystic kidneys. The extreme immaturity of the organs under examination makes the value of these observations very doubtful. If such structures were to be identified in older kidneys the proposition might be worth attention but, as Busse (1899) remarked many years ago, such "cell rests" have never been convincingly demonstrated.

If the embryonic hypothesis is not satisfactory, what other explanation can be offered for the specific pattern of age incidence? A reasonable approach might be along chemical rather than cellular lines. The chemical environment of growing kidney cells may possibly favour this particular kind of tumour, an explanation in line with "organiser" concepts of modern embryology (Needham, 1942; Muir, 1951). Admittedly this is surmise, but it serves to show that the age pattern can be explained in more than

one way, and that its histogenetic significance is more difficult to interpret than appears at first sight.

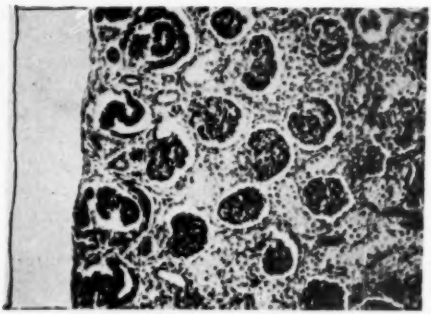

FIG. XII. Cortex of kidney of new-born infant. Note the persistent subcapsular nephrogenic zone still forming glomeruli.

All this leaves the embryonic hypothesis in a shaky condition, and we must now consider whether it can be buttressed by further histological study. It has been shown already that the histology of the childhood group strongly supports a stem-cell hypothesis stated in general terms. However, this conclusion has not been universally accepted in the past. Many investigators from Wilms (1899) to Masson (1938) have tried to derive each histological structure of the tumour from its corresponding embryological counterpart. The epithelial elements have been assigned to renal blastema, cartilage and bone to the sclerotome, muscle to the myotome and nerve fibres to the neural crest. The variety of these structures has been the main problem, as it is difficult to see how they all could come together to form one tumour. No space need be wasted on the ingenious hypotheses of "inclusion" and "illegal mixture" which were produced to meet this difficulty. The whole proposition was obviously absurd, and another body of pathologists set to work on the general theory that tissues developed by differentiation from a stem cell within the tumour itself. The first approach to this idea was made by Muus (1899), who squarely stated that the tumour components arose from renal blastema. According to this idea the epithelial structures in the tumour are the only ones breeding true to type, and others — striated muscle, cartilage, and bone — being due to metaplasia. This hypothesis has received strong backing from Ewing (1919) and Willis (1948), and may be regarded as the modern concept.

It may be modern, but it still fails to convince. We may smile with conscious superiority at the rigid way in which our predecessors attempted to derive like from like, but at least it was logical within its limits. The trouble is that as soon as one admits that a tumour is not derived from some normal counterpart, but rather by metaplasia from something different, the whole histogenetic house of cards collapses and it becomes impossible to derive with any certainty the origin of a tumour from its histological appearances. The renal blastema hypothesis does not face the issue squarely. It selects the epithelial structures, says they are truly derived from an embryonic blastema, and dismisses everything else as metaplastic. I would submit that there is no reason to suppose that epithelial structures are in any way different from others in this regard and that the appearance of these structures cannot give any clue to their origin. This means that identifications such as "pro-glomeruli" or "primitive tubules" may be true but equally well may result from reading too much into histological resemblances. For these can be misleading, as proved by the occasional appearance of "pro-glomeruli" in tumours remote from the kidney. They have been observed in prostatic carcinoma, fibrosarcoma (Bosse, 1940), and synovioma (King, 1952, Fig. XIII). In this last situation they can hardly be derived from renal blastema except by an "illegal mixture" of truly prodigious dimensions!

At this stage the only course left to us is to jettison our theories and admit our ignorance. The observations support a stem-cell hypothesis in general terms but give us no indication of the origin or nature of the originating cell. There is no proof that it is embryonic; it may even be mature. We simply do not know. What is more, observational methods alone cannot, by their very nature, help us further.

Fortunately the matter does not end here. There is still the experimental approach which has already gone far to explain the origin of another type of mixed tumour, the teratoma. The production of this tumour

following injection of zinc chloride into the testes of cocks has led many to conclude that it can arise from adult testicular cells (Michalowsky, 1926; Bagg, 1936; Ferguson, 1937). I suspect that future experimental work on mixed renal tumours may lead to a similar conclusion. For the moment, the results of experimental carcinogenesis in the kidney are meagre, being apparently confined to the production of malignant adenoma by stilbene derivatives (Vasquez-Lopez, 1944; Matthews, Kirkman, and Bacon, 1947; Kirkman and Bacon, 1950). As there seems little doubt that the fundamental problems of origin can only be solved along experimental lines, the scantiness of these reports provides a challenge to further investigation in this field.

A final plea about nomenclature. Let us scrap eponyms like "Wilm's tumour" and embryological abominations like "metanephroblastoma" or "mesoplastic nephroma." Why not instead use the simple descriptive term "mixed renal tumour" for the whole group, adult and juvenile? At least by doing so we would admit how much we do not know; and such an admission is the beginning of wisdom.

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SURGERY IN ULCERATIVE COLITIS

By Sir Albert Coates

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THE use of surgery in the treatment of patients with ulcerative colitis is comparatively recent. Appendicostomy and washouts were tried thirty years ago. Ileostomy became more fashionable in the mid 30's—complete side tracking of the intestinal flow being found to cause improvement in these patients. Relapse, however, occurred and colectomy was found to be the only certain means of eliminating the toxaemia, haemorrhage and debility.

The experience of the average general surgeon, in the treatment of this disease, must necessarily be limited, hence the value of reports of even small series of patients which have been followed over a period of time.

The treatment of ulcerative colitis is a judicious combination of medicine and surgery. In the last sixteen years (eleven of which were in Australia) I have had about fifty patients suffering from this disease—confirmed by sigmoidoscopy and radiography. They were of the moderate and severe grade. Thirty-five were subjected to surgery after various forms of medical treatment had been carried out. The oldest was 54, the youngest 12 years of age. The average age was 31 years. The average duration of this disease was 8 years (15-3); 11 patients were males and 24 females.

Many of these patients had had long periods in hospital, up to two years, and only when the physician-in-charge was satisfied that the disease was beyond medical control, was operation undertaken. An additional two patients were operated on by me at the request of surgeons in England in 1950. The remainder were referred back, after consultation, to their physician for continued medical treatment.

Even though there was gross change in the colon, as shown by X-ray and sigmoidoscopy, if the patient could lead a reasonable life without too much inconvenience, suffering only temporary remissions, I have advised continuance with medical care. An example:

A business executive in 1952—a man of 48—had had the disease for 10 years; he passed a little blood and loose stools intermittently—especially when worried at his job, 4-6 stools a day. Sigmoidoscopy showed a small ulcer of the rectum, oedema of the mucosa. He was kept fairly comfortable on medical and dietetic care. Routine check up of rectum and barium enema was done. He is an insurance manager—at present he prefers to remain as he is rather than risk a permanent ileostomy or an ileo-rectal anastomosis with the 50-50 chance of still passing 6 stools a day.

A boy of 20, whose father died and mother married again, had typical ulcerative colitis. Under psychiatric and medical treatment he improved a great deal and leads a reasonably comfortable life.

Thus, a diagnosis of ulcerative colitis by the surgeon does not necessarily indicate operation. Other conditions should also be considered:

(a) Melanosis coli, which is not a surgical condition;

(b) procto-sigmoiditis, which responds to medical care.

My first patient, treated surgically, was a single woman of 30, who had been treated medically for some years. Appendicostomy had failed. In 1938, I performed an ileostomy. She improved very much but began to deteriorate in 1939. I then did a total colectomy, leaving the rectal stump.

She presented in 1947, with a fibrous, pus-containing bag which was her rectum. As she was a little below par and wished the removal of the septic remnant, I excised the rectum.

She has since enjoyed excellent health. The permanent ileostomy is a small aperture which discharges a semi-solid substance of toothpaste-like consistency, usually after each meal. Obviously, the ileum has taken over the water absorbing function of the colon.

In 1946, I attempted to join the ileum and the rectum by direct anastomosis in two patients. One died of peritonitis and other complications. Since then, I have never attempted that procedure but have either brought out the ileum and proximal end of the recto-sigmoid stump together—or, more recently, have closed the rectal stump as low down as possible after excising all the sigmoid colon and leaving the ileostomy by itself. A later operation has then been done if and when an ileo-rectal anastomosis was feasible.

Surgery is not wthout its worries in these cases — complications can be troublesome; and a wise surgeon does not readily embark on operative treatment of this disease without close collaboration with the physician.

The following are the conditions for which operation has been performed:

1. *Perforation, fistula formation*

(a) V.F., female, aged 19, had had a perforated caecum. She had been under treatment (medical) for 2 years. At operation, ileostomy was performed with drainage of peritoneal cavity. Later, under streptomycin a two stage colectomy was performed and 5 years later an excision of rectum.

(b) Mrs. M., aged 29 years, had a recto-vaginal fistula and also a fistula opening just above left popliteal fossa. Ileostomy had been done elsewhere. Total colectomy-proctectomy cured her. She is now very well.

2. *Repeated severe haemorrhage*

Mrs. P., aged 29 years, had had repeated severe bleeding which necessitated ileostomy in 1945. Excision of the colon was performed in 1948 (A.E.C.). Her rectum was not severely ulcerated and the valves were elastic. Ileo-rectal anastomosis was carried out by the Mickulicz-Devine technique. A year later, she was passing only two stools a day. A small incisional hernia was then repaired.

In 1954, following domestic worry — infidelity of husband—there was diarrhoea with some blood and ulcers were seen in the rectum. She was treated medically and she recovered with only 3 stools a day. In March, 1955, she was very well with 2 stools a day and no blood. Rectal mucosa was red but the valves were normal. This case illustrates that it is possible to obtain a good result with ileo-rectal anastomosis, but that the rectum is vulnerable and needs to be observed, especially under condition of emotional stress. In July, 1955, she is 5 months pregnant; she is very well. Sigmoidoscopy shows the rectum to be the same as previously.

3. *Wasting — general deterioration despite medical treatment*

Miss J.S., aged 13 years, had diarrhoea—9 stools a day for four years. She was in hospital most of that time, in Sydney and Melbourne. There was blood and pus in the stools constituting a very offensive discharge. On sigmoidoscopy, the mucosa was spongy and prolapsed easily into instrument; no ulcer was seen. On X-ray colon was ribbon-like; incidence in the caecum was severe. Ileostomy was performed. 4th August, 1948, there was great improvement. 23rd September, 1948, colectomy including 18″ of lower ileum was performed. Lymph nodes in the ileo-caecal angle were very large; the caecum had perforated extra-peritoneally. An ileostomy with a double-barrelled alignment of ileum and lower sigmoid stump was carried out. Pathological examination showed that the lower ileum was involved in ulceration and thickening but that the main incidence was in the caecum and ascending colon. 1st November, 1948, the spur was crushed. 22nd February, 1949, she was very well and had gained 2 stone in weight. There were 5 stools in 24 hours; she was attending school. 11th May, 1951, she never needs to leave class now and she is good at all sports. 6th June, 1955, she had pased all her examinations. There were 5-6 stools in 24 hours; her weight was 10 stone 7 lbs. On sigmoidoscopy, the mucosa was pale. There were not any ulcers. This patient illustrates Brooke's type of ileo-colitis—the main incidence of the disease being in the right side of the colon and ileum. Although the left side of the colon was also involved, the rectum was relatively normal.

Mr. H., aged 54 years. He was referred to me in 1947 by Dr. L. E. Hurley, with 3½ years history of diarrhoea with blood in the stools (6 stools a day). 16th February, 1948, ileostomy was performed. 16th March, 1948, colectomy was carried out—double-barrelled ileum and sigmoid anastomosis. Following this there was some electrolyte inbalance. blood chlorides being 400 mgm. per cent. and he was very sick. 3rd November, 1948, the spur was crushed and anastomosis completed. There were 8 stools a day. An abscess in the left iliac fossa was opened and a fistula formed. There was erosion of skin round the ileostomy. 10th September, 1951, excision of all of the sigmoid colon and end to side anastomosis of ileum and rectum was performed. 7th January, 1952, there were 5-7 stools a day but the patient was very well. In 1955, he is now quite well and has gained 3 stone in weight.

In four other patients this same anastomosis has been done.

These patients presented serious problems and an attempt was made to achieve comfort for the patient whilst at the same time removing the bulk of the disease. I could find no evidence, in 12 patients on whom I had done ileo-sigmoid or ileo-rectal anastomosis, that the ileal contents improved the condition of the rectum. Nor could I mobilize the rectum and exteriorize it as described by Devine. The perirectal oedema and stiffening of the rectum precluded such immobilization. In one case the ileo-sigmoid anastomosis was a failure and I excised the remaining sigmoid

colon (see above). In another, recurrence of the disease in the lower part of the ileum or a spread from the rectum necessitated the remaking of an ileostomy. Severe erosion of the abdominal wall, uncontrollable by all methods, made life so unhappy for this 24-year-old boy that the further excision of diseased lower ileum was done and the rectum anastomosed to it by the end to side method. This patient, first operated on by me in 1946, had a large carcinoma of the transverse colon. He was very well in 1953 and came to thank me for helping him. A few weeks later, he died in a country hospital from a perforated duodenal ulcer.

In 8 patients, the rectum was removed at intervals from 3 months to 7 years after colectomy-ileostomy. In all of these patients, the rectum showed no sign of improvement; in two, it was so bad that, though separated in time because of the low state of the patients, its excision formed part of the primary procedure. They both had recto-vaginal fistulae. If a patient is in such good condition that colectomy and rectal excision may be done in one stage, it is my view that the rectum may be worth retaining or, alternatively, that the patient may not need such radical treatment. The exception to this is the case of bleeding polyposis associated with the colitis. Two such patients both died: one of suppurative pylephlebitis, the other of mesenteric thrombosis. Four other patients died: one, an emaciated young woman who had been treated with cortisone, after ileostomy in England. She drained her body fluids away through her ileostomy and was uncontrollable biochemically. One case died of small bowel obstruction after crushing of the ileo-sigmoid spur, one from peritonitis and subphrenic abcess and one from broncho-pneumonia and flooding with intravenous fluids. Thus three of these deaths were preventable; they should not have occurred.

Nine remaining patients have had a colectomy and ileostomy and retain their rectums, but I am certain that unless some dramatic medical discovery is made soon they will almost all have permanent ileostomies. They are well and content to be watched, hoping for the day of return to normal.

In addition to the patient who died of obstruction, five others have had to be operated on for adhesions causing obstruction to the small bowel. The usual site is to the stump of the meso-colon—the infected glands are cut across during colectomy and form tiny abscesses and loops of small gut adhere to them. The colic gutters, whether peritonealized or not, do not seem to cause trouble. The correct anchoring of the ileostomy and the closure of any window or noose is, as Brooke remarks, essential. I have not had trouble from that cause.

Other post-operative complications are:

(a) After ileostomy—

Prolapse of ileostomy	2
Stenosis of ileostomy	1
Haemorrhage	1
Intractable ulceration of abdominal wall	1

(b) After colectomy—

Incisional hernia	2
Femoral thrombosis	1

All the twelve patients with ileo-rectal or ileo-sigmoid anastomosis are well. One is a nurse, another a Red Cross worker and all carry on their normal work. Two have had relapses of frequent stools and a trace of blood (both females); they will need regular rectal inspection. Carcinoma is a distinct risk. All the eight patients who have had rectal excision are well. Of the nine patients who retain their rectum, I expect most to have a permanent ileostomy. Of the six who died I think four, had they survived, would probably have had a permanent ileostomy.

So 60 per cent. of my cases, roughly, could qualify for permanent ileostomy. Maybe some of the 12 who have been closed will, later in life, be happier with a permanent ileostomy. The idea of a permanent ileostomy becomes less terrifying as time passes.

The parents of the patient, V.F., were very worried about the girl having a permanent ileostomy. They implored me to save the rectum and join the ileum to it. I resisted this temptation and, with the help of many other opinions, both of physicians and surgeons (none of whom differed from my own), the girl welcomed the removal of the rectum and lower sigmoid colon. She has adapted

herself to the new stoma over 5 years and, like the one-legged man, was no longer a self-pitying patient.

These facts lead me to believe that the treatment of the disease is as important as the treatment of the patient and that radical surgery will be the proper procedure in patients with extensive involvement of the whole colon and rectum but that less radical measures, that is to say, colectomy and ileo-rectal anastomosis are feasible in those patients who retain a rectum which, if not normal, is one which can be regarded as an elastic container with a reasonable mucous lining. An abdominal ileostomy is preferable to an uncontrollable anal ileostomy.

The ideal surgical procedure, in my view, is to do a colectomy at one sitting, making an ileostomy at the same time, closing the rectal stump and, if necessary, removing it at a later operation. In this series, it has been done four times. I have regarded the other patients as too ill for such an attack and have preferred to do a preliminary ileostomy, followed by colectomy later.

With better medical preparation and with co-operative physicians who present fewer moribund patients for surgery, I believe that colectomy will be the procedure of choice as a primary measure. With good team work, a bad rectum may be removed at the same time. However, staging of operation is an old surgical trick and may prove invaluable to the less experienced in the art, as well as life saving to the patient.

VEIN GRAFT FOR ARTERIAL INJURY

By Barton Venner

Adelaide

IT is not common to see a case of acute ischaemia of the upper limb; nor is it common to be able to ameliorate, by early surgical intervention, such a condition.

The case to be described is that of an injury to the arteries in the cubital fossa where, it would usually be thought, no major peripheral circulatory defect should be produced. However, the circumstances of the injury were such that the loss of at least part of the hand was at first anticipated.

Case History

B.L., a lad of 16 years, was admitted on New Year's Day, one and a half hours after a motorcycle accident, in which his left arm was severely lacerated in the cubital fossa. There were no other serious injuries and the condition of shock responded readily to general measures.

The left cubital fossa had been opened by a large laceration forming a flap with its apex on the biceps brachii muscle, and its base encircling the mid-forearm for all but two inches. There was little muscle damage but the occluded stump of a large artery could be seen pulsating.

The forearm was cold — a pallid blue colour, anaesthetic and immobile from about the middle of the forearm. Arterial pulsation was impalpable distally.

At operation, two hours after admission, the *pronator teres* and *brachioradialis* muscles were found to be lacerated, but other groups were largely intact. The radial and ulnar arteries had been severed about half an inch below their origins, and the distal end of the ulnar artery was not found. The stumps of vessels taken to be the common interosseous, radial recurrent and ulnar recurrent arteries were found on the proximal vessels, and it appeared that most of the distal connections for an anastomosis around the elbow, as well as some proximal muscular branches, were lost. The median nerve had been exposed, but it was thought that its anterior interosseous branch had been avulsed.

After excising the damaged muscle, a piece of polythene tubing was used to join the brachial and radial arteries. This produced a dramatic improvement in the circulation to the hand, with brisk venous bleeding from distally. Accordingly, a segment of the cephalic vein was exposed by a short extension to the upper part of the laceration, and the segment was reversed and sutured in as a graft between the radial and brachial arteries, which were trimmed to receive it. The skin was closed over the graft, with drainage, and a plaster splint applied.

The initial circulatory improvement was not fully maintained after twelve hours, when the greater part of the arm was cooler than normal and cyanotic in colour, and it was feared that the graft had become occluded. But, in a further twelve hours, the colour had returned progressively to normal and finger movements were occurring.

The viability of the whole limb now remained in no doubt, but over the next few days extensive gangrene of the skin flap developed and a strong tendency to clawing of the fingers necessitated splintage.

On the nineteenth day, at a second operation, a large flap of gangrenous skin was removed from the cubital fossa and upper forearm (the vein graft or main arteries were not seen). The exposed parts of the *flexor carpi radialis, palmaris longus* and *flexor digitorum sublimis* muscles were seen to be pale in colour, firm and non-contractile, and did not bleed on cutting; their tendons were already adhering to the surrounding connective tissue. These muscles were excised as completely as possible, leaving a good length of the median nerve exposed. What was visible of the *flexor digitorum profundus* muscle looked unpleasantly pale and avascular, but it seemed undesirable to excise further tissue.

At several subsequent operations, the large granulating area was covered by split skin grafts.

The outcome of the next four months of persistent physiotherapy was a very useful hand. There was moderate scar contracture of the elbow joint with recurrent ulceration due to stretch. Wrist flexion and extension occurred over a useful range. The thenar muscles were contracted, and limited opposition of the thumb but the *flexor pollicis longus* muscle was active. Except for the index, the fingers could be flexed well at the metacarpo-phalangeal joints, and slightly at the interphalangeal joints and the bad features of clawing were absent.

The index finger had no power of active flexion in the interphalangeal joints. Passive joint movement was good.

It was considered that further improvement could be gained by:

(i) Scar excision and skin graft to the cubital fossa.

(ii) A tendon transplant for index finger flexion.

(iii) Fixation of the thumb in opposition.

DISCUSSION

Division of the brachial artery at the elbow only exceptionally results in loss of part of the limb (Medical History of World War I, 1947).

The attitude of the hand which occurs in Volkmann's ischaemic contracture (Volkmann, 1881) is due to necrosis and contracture of the long muscles of the forearm, resulting in extreme flexion of the wrist and inter-phalangeal joints, with hyperextension of the metacarpo-phalangeal joints.

It is considered that arterial insufficiency occurred in this case because the injury resulted not only in division of the radial and ulnar arteries but also in stripping of the collateral connections and muscular branches in the upper forearm. Restoration of continuity by vein graft resulted in a sufficient flow of blood through to the hand, but insufficient to the forearm. Excision of part of the contracting forearm flexor muscles helped to prevent the development of a contracted hand.

The history has a sequel. At a subsequent operation for excision of scar tissue and grafting to open the flexure of the elbow, the first incision at the level of the former graft entered directly a large thin-walled vessel embedded superficially in the scar tissue. Profuse arterial bleeding occurred. When controlled by fingertip pressure over this vessel, the hand became pale and mottled.

Resuture of the incision in the vessel was carried out without any permanent ill effect. Three weeks later, this vessel could be felt pulsating distal to the incision, and pallor and paraesthesia of the hand developed within seconds after it was occluded by one finger.

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TRAUMATIC PARAPLEGIA

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IN this paper cases of paraplegia due to thoraco-lumbar injuries only will receive consideration. Fractures and fracture-dislocations of the cervical spine are omitted as they usually cause some form of quadriplegia.

There was a time when most patients afflicted with paraplegia from injury to the spinal cord were doomed to total invalidism. If they survived the initial onslaughts of bedsores, acute urinary infections and pneumonia, they were obliged to spend the rest of their days (often fortunately not very many) in bed in a hospital or home for incurables, a burden to themselves, their family and the community. Now, with the advent of the sulphonamides and antibiotics, and with improved facilities and equipment for nursing, the early dangers of the acute stage should never arise or, having arisen, should be readily brought under control. Then, with adequate mental and physical rehabilitation, it is possible for these patients, once they have mastered the "activities of daily living" to lead useful and happy lives.

Treatment and rehabilitation of this type cannot be haphazard affairs. They must be carefully planned from the outset. To discuss the treatment of traumatic paraplegia as the healing of bedsores, the overcoming of severe urinary infections, the correction of spinal deformities and the mobilization of stiff joints, is out of date. To avoid these complications is the aim and this requires meticulous care and nursing which can only be achieved in a special centre staffed by a team properly trained for this class of work and interested in it. Such a team should be headed by a full-time director with one or more assistants, nurses (male or female), physiotherapists, occupational therapists, social service workers and industrial officers, whilst the services of visiting neurosurgeon, orthopaedist, urologist, and plastic surgeon are essential.

A few well-known centres and teams have already played a notable part in this work during and since World War II, in Britain, Canada and the United States, not only by results obtained on their own patients but also in setting a standard for others by showing what can be accomplished. In these Centres, however, most of their energies have been taken up in repairing the bad results of early treatment given the patients who have only been admitted weeks or months after the injury. To be really effective, a spinal centre should be able and willing to accept any paraplegic patient at once. It should be an emergency centre and always have a bed available for an acute case.

In the State of New South Wales no such centre, as yet, exists and all cases must be dealt with in general or private hospitals with, as a rule, shocking results.

In this respect the experiences published recently from a Spinal Centre at Sheffield are interesting. This Centre has 50 beds which have been found sufficient to deal with paraplegia occurring in a heavy industrialized area of about two million persons. Since its establishment 150 cases have been admitted and of these 71 were acute cases received on the day of injury or very shortly afterwards. In none of these cases did any serious bedsore or urinary infection occur. All the patients with dorso-lumbar lesions have been up and out of bed in 3 months and discharged home in an average of 9 months, whereas, in the late cases, the period of hospitalization averaged two years.

General Considerations and Early Diagnosis

As soon as a case of traumatic paraplegia is seen, and in order to determine the probable nature of the resulting damage to the spine and cord, it is important to obtain an account of the nature of the accident with special reference to the lines of force and mechanics involved, that is, hyper-flexion, hyper-extension, compression or torsion. As each variety of injury will cause a different type

of fracture or fracture-dislocation, it will call for a different first-aid method of transportation, for example, prone or supine positions with supports.

The next step is to arrange for accurate and good technical skiagrams to be taken on a Potter-Bucky diaphragm or with a Lysholm grid, before the patient is put into a bed, in order to determine the exact nature of the damage sustained by the affected vertebrae, that is, fracture, dislocation or both and especially, if there is a dislocation present, whether the articular facets are inter-locked or not.

Information obtained from neurological examination

While these films are being developed a careful neurological examination should be made and the findings recorded to determine, as soon as possible, the clinical level of the damage to the cord. This may not correspond with the anatomical and X-ray level, the disparity being due to the presence or absence of injury to nerve roots.

The nerve fibres in the spinal cord have no power of regeneration and thus transection of the cord, either anatomical or physiological, is an irrecoverable lesion. On the other hand, nerve roots are peripheral nerves. They are more resistant to injury and are capable of regeneration after transection. Thus cord and root lesions are very different and it is important to distinguish the one from the other as, generally speaking, the prognosis of root paralysis is better than that of cord paralysis; and the final results differ in that, with a cord transection, there is paralysis of the upper motor neurone type below the level of the lesion and return of local reflex activity; whilst with anatomical division of nerve roots, if recovery does not occur, the paralysis is flaccid and there is no return of reflex activity in the affected segments.

The spinal cord ends at the lower border of the first lumbar vertebra. Below this level the spinal canal contains the *cauda equina*, whose components are morphologically the same as peripheral nerves.

The first lumbar neurological segment lies nearly opposite the body of the 10th thoracic vertebra; and the first sacral segment lies opposite the lower border of the 12th thoracic vertebra. Thus, all the lumbar cord segments lie between T10 and T12 vertebrae; and all the sacral segments lie between the lower border of T12 and lower border of L1 vertebrae. The spinal canal, therefore, at the thoraco-lumbar junction, contains the cord and all the lumbar nerve roots passing down from their segments of origin to their proper intervertebral foramina.

Above the level of the 10th thoracic vertebra, the nerve segments and roots get more and more nearly opposite their corresponding vertebral bodies.

There is one other important anatomical point to remember about the blood supply of the cord. In the embryo, the longitudinal spinal arteries are reinforced by arteries which enter along each nerve root; but during development all except four or five of them disappear. One of the remaining vessels is fairly constantly found running in the 2nd lumbar nerve-root and therefore passing across the thoraco-lumbar vertebral junction.

In addition to the above considerations, the spinal canal in the upper and mid thoracic regions is very narrow and less mobile than in the thoraco-lumbar regions, so that lesions above T10 vertebra usually result in a severe grade of compression with or without dislocation which causes a complete transection of the cord and nerve roots at almost the same level, with permanent paraplegia and with the neurological and X-ray lesions at practically the same level. A lesion between T12 and L1 vertebrae may injure the cord at the S1 segment, but the neurological level may be as high as L1 due to the nerve roots from all the lumbar segments being involved. Below L1 vertebra, the lesion only affects the nerve roots of the *cauda equina*.

In actual practice, the cord level and the vertebral level of damage may not always correspond, as traction and vascular disturbances may result in a higher level of cord transection. Often, however, it can be shown that the paralysis and anaesthesia above the level of the vertebral damage are due to a root lesion alone. Accordingly patients with thoraco-lumbar injuries may be divided into four groups:

(a) Cord divided: complete escape of nerve roots; neurological and radiological levels coincide.

(b) Cord divided: partial escape of nerve roots; an irregular pattern of lumbar segmental paralysis with complete paraplegia in the sacral segments corresponding with the radiological level.

(c) Cord and all roots divided: a complete lumbar and sacral paralysis with a radiological level at the 5th lumbar or 1st sacral segment.

(d) Cord divided and some destruction of the grey matter of some lumbar segments, due to traction or interference with the blood supply above the radiological level by thrombosis of the artery with the 2nd lumbar nerve root; this results in sacral cord isolation and upper motor neurone type of paralysis with permanent complete lumbar segmental paralysis of a lower motor neurone type.

Estimation of damage to the cord

Having determined, from the history and neurological examination, the clinical level of the cord injury and whether nerve roots have been damaged or not, it is important to try to determine whether the cord has been transected or not. Complete interruption of all impulses, together with total suppression of distal segmental functions, may follow cord injury without transection. This suppression is often wrongly referred to as "spinal shock". Spinal shock is really a total suppression of function in those segments of the cord below the level of transection and, in human beings, it may last for weeks. It ultimately disappears and the isolated segments regain reflex functions. And as in upper motor neurone lesions the paralysis which was flaccid, becomes spastic.

The similar picture of immediate loss of function in a cord not transected should be called "spinal concussion". Spinal concussion also disappears in time but with the return of normal function — motor power, sensation and reflexes. Spinal concussion alone, occurring in a non-transected cord, will usually show some signs of recovery within hours. Indeed, the motor and sensory loss may never have been absolutely complete and some slight continuity of motor power or sensation may be noted on the first examination.

Partial lesions of the cord, when examined always have some sign of continuity such as weak motor power or perverted sensations or both in one or more segments but although full recovery may occur in these segments, it may not extend to other parts of the cord. Thus, unless some continuity can be demonstrated by the return of some function below the level of the lesion in a matter of hours, it can be presumed that the cord has been transected and is unlikely to recover.

In spinal concussion, all reflex activity is usually absent below the level of the lesion; thus the preservation of reflex activity in segments below the lesion, without any sensation or motor power, is almost certain evidence of cord transection.

Some little time ago, the use of a substance called *Piromen* was advocated in cases of paraplegia, especially where the roots of the *cauda equina* were involved. It is a bacterial polysaccharide which is given parenterally to promote, by increasing phagocytosis, a generalized antifibrotic and anti-glial reaction. It was said to dissolve the glial reaction in the cut ends of peripheral nerves and to allow regenerating neurones to send their axones more readily across the damaged area. It may be given along with DOCA but its use is still in the experimental stage.

Information obtained from skiagrams

Bearing these considerations and the mechanism of the injury in mind, the skiagrams must now be carefully studied to determine the exact level of the injury and whether there is any deformity. Deformity may be due to fracture or dislocation or both, and may result in a stable or unstable spine.

Stability of the spine after injury depends not upon the severity of the fracture of the vertebra but on the degree of damage to the posterior ligaments. If the posterior ligaments remain intact they act as a fulcrum and a typical crush fracture results. The fragments are impacted; the articular facets are intact and the spine is stable in its deformed position. Such injuries only rarely result in partial cord or nerve root lesions.

If the posterior ligaments rupture, the fulcrum is lost and a pure dislocation often results with locked articular facets. Torsional

violence with rupture of the posterior ligaments and fracture of one or other articular process allows the upper vertebra to swing upon the lower. The intervertebral disk and a slice of the upper part of the lower vertebra are carried with the upper vertebra resulting in a very unstable fracture-dislocation, which is the common cause of paraplegia, the cord and nerve roots being crushed between the lamina of the upper vertebra and the body of the lower vertebra.

This type of lesion can be recognized by palpation of a gap between the affected spinous processes and the lateral shift of the lower one. However, when it occurs below the level of the 1st lumbar vertebra, extraordinary deformities may be found without irreparable neurological lesions of the nerve roots.

Thus, from the history, careful neurological examination and study of satisfactory skiagrams, a clear picture of the lesion should be obtained. Distinction between cord and root lesions can be determined and transection or partial damage of the cord recognized. The spinal injury can be assessed as stable or unstable.

IMMEDIATE MANAGEMENT

The objects of the early treatment in cases of paraplegia are threefold:

(1) To make sure the cord and nerve roots are free from any possible compression which may hinder spontaneous recovery or result in further damage.

(2) To restore the alignment and stability of the spine, as far as possible, to allow for future weight bearing.

(3) To make nursing easy and adequate so that bedsores, urinary infections, pains and reflex spasms may be avoided.

(1) Relief of compression of spine and nerve roots

Relief of compression may have occurred spontaneously when the patient is first placed in the prone or supine position and very little deformity may be found on clinical examination or by X-rays. Even so, and despite the impression that the cord may have been transected, lumbar puncture and the performance of a Queckenstedt test should always be carried out as soon as possible to make sure there is no block from extra- or intra-dural or even intra-medullory haemorrhage, or from an extruded intervertebral disk. If a block be demonstrated, then it should be confirmed 12-24 hours later and if still present, exploratory laminectomy proceeded with immediately. If no extra-dural lesion is found, the dura should always be opened carefully and the cord examined. If not transected, it should be needled for haematoma or even incised in the mid-line and decompressed, by leaving the dura open.

This plan of campaign is particularly indicated where the paralysis and anaesthesia are due to lumbar root lesions; the nerves must be freed from pressure and protected from further injury if recovery is to take place. The lumbar roots are very important as they control flexion and some extension of the hip, extension of the knee and much of the sensation of the leg.

When there is a deformity of the spine, either due to fracture or fracture-dislocation, it must be reduced, even if the cord appears to be transected. Reduction can often be affected by manipulation in extension between two tables, but if there is a dislocation with interlocked or displaced articular facets, open operation must be undertaken. Queckenstedt tests are then performed, as outlined above, to make sure that there is no compression of cord or nerve roots. If reduction is complete and there is still evidence of compression, then myelography and possibly further operation would be justifiable.

(2) Restoration of alignment and stability of the spine

If the vertebral injury is of the stable type and the canal not grossly deformed, then reduction and support are unnecessary; if the Queckenstedt tests are normal, exploration is contra-indicated.

If the alignment of the spinal column is so badly upset that stability is lost and there is a risk of further damage during the necessary turnings for adequate nursing, then steps must be taken as soon as possible to restore the alignment, or at least to stabilize the spine, at once and almost as an emergency.

Even if there are only few or no signs of cord or nerve root involvement, such steps are necessary, so that the patient can be

nursed properly and turned frequently, without fear of further damage, and so that the erect or sitting position may be assumed as soon as possible to avoid urinary tract complications. It is surprising sometimes what severe deformities can exist with few or no neurological signs.

Reduction by manipulation and support by external splinting are often disastrous. Plaster beds should never be used as they lead to large and very chronic bedsores. Thus, it is probably safer to affect an adequate reduction of the deformity by open operation and to maintain this reduction and stabilize the spine by internal fixation with screws, or with plates bolted to the spinous processes.

(3) Adequate nursing

(a) Avoidance of bedsores

In the early stages, and when two-hourly or even hourly turnings may be required, the use of a Stryker Rotoframe type of bed is advised. This can be carried out by one nurse, if necessary. Otherwise a Dunlopillo mattress should be used on top of fracture boards on any ordinary bed and the patient should be nursed in the prone position as much as possible.

Oedema of the back, buttocks and lower limbs often occurs, due to lack of sympathetic vaso-constrictor impulses in the regions below the damaged level of the cord and is the precursor of decubitus ulcers. It can often be prevented, or relieved, by elevating the foot of the bed or frame on blocks or other supports.

Often in the early stages there is an acute loss of weight due to excessive nitrogen metabolism; adequate nutrition must be maintained by giving a high protein, high vitamin diet, along with, when available, daily short exposures to sunlight.

(b) Avoidance of urinary infection and retention

Infection usually occurs following retention of urine, with over-distension of the bladder and the frequent use of a catheter. Under wartime conditions, an early supra-pubic cystostomy is justified and provides a simple foolproof method of bladder drainage needing little attention, whilst the patient is being transported, often over long distances and being moved frequently; but it allows the bladder to shrink and there is always some leakage of urine which irritates the lower abdominal wall and keeps the perineum moist, thus favoring the development of bedsores. Similarly an indwelling catheter, draining direct to a "Winchester" bottle beneath the bed is bad as, despite daily irrigations and stretchings, the bladder soon becomes constricted and fibrotic.

By far the best procedure is to insert some form of self-retaining catheter and connect it to one form of recognized tidal drainage apparatus. The Cone and Bridger's modification of Munro's Apparatus has a built-in cystometer, is simple and reliable and is recommended for routine use.

The irrigating fluid may be Allbright's solution G, with or without $\frac{1}{2}$ per cent. acetic acid or $\frac{1}{2}$ molar buffered citric acid solution with pH on the acid side to prevent the formation of phosphate calculi.

If the lesion is above the *conus medullaris*, the patient should develop a "reflex" bladder which will empty at regular intervals if given an adequate stimulus and if manually compressed, and then tidal drainage is no longer required.

But, in lesions of the *conus medullaris* or of the *cauda equina*, retention may persist or painful spasms may occur in a hypertonic "neurogenic" bladder. Then, relief may be afforded by severing the pudendal nerves and by trans-urethral resection of the bladder neck. In some more obstinate cases, total removal of the anterior and posterior nerve roots from T12 through to L5, or destruction with intrathecal alcohol, has produced an "automatic" bladder which will completely empty itself at routine times.

It has been proved conclusively, that lengthy periods of decubitus in the supine or even prone position, will result in the formation of renal and bladder calculi; thus the assumption of the erect position in a wheelchair or standing for certain periods of time each day, must be attained as soon as possible.

(c) Relief of pains

Despite the absence of all sensations below the level of the lesion, pain of an intractable burning nature is often felt, referred to the

portion of the body rendered analgesic. The nature and severity of this pain is assessed by the patient in the light of past experiences and emotional stability. Certain patients are able to endure it and lead useful lives; others cannot cope with the problem and suffer so severely that surgical treatment is required if survival and rehabilitation are to be made possible. The explanation of such referred pain is not obvious but it is supposed to arise from the fibres in the pain-carrying tracts of the cord, just above the transected level; thus its occurrence is more common in traumatic and especially war-wound cases.

In a series of 125 cases of traumatic paraplegia, treated in the Neurosurgical Units of the Department of Veteran's Affairs and at Lyndhurst Lodge at Toronto, Canada, 38 cases (30 per cent.) had disabling pain but only 9 cases (7 per cent.) required surgical relief.

Bilateral high thoracic spinothalamic tractotomy would appear to be the procedure of choice, but occasionally bilateral prefrontal lobotomy may be necessary.

(d) Relief of reflex flexor spasms

Involuntary spasms may occur with any type of paraplegia once the cord has regained its autonomous function, but this is usually with lower thoracic or upper lumbar lesions. They come with the onset of spasticity and are provoked by touch or painful stimuli applied to any portion of the body below the affected level. They may be very severe and distressing, make nursing difficult, prevent the patient from gaining his independence and disturb sleep.

Anterior rhizotomy, from T9 or 10 to S1 bilaterally, was first proposed and practised by Munro but his is a major procedure and could not always be carried out with safety in debilitated patients.

Others advocate neurotomy of the peripheral nerves to the most powerful flexors and adductors of the thigh, that is, the obturator and part of the femoral nerves, whilst the hamstring muscles and tendons and sciatic nerves are cut in the popliteal fossae.

A third and simpler method of abolishing spasm is by subarachnoid injection of alcohol (about 15 ccs.). With the patient placed in the supine position, the alcohol rises towards the anterior roots in the caudal portion of the sac and the skeletal muscles of the lower abdomen and lower limbs are rendered flaccid. The bladder may become atonic, too, so that this procedure should not be done on patients with automatic bladders which function well.

Recently, in some very severe and intractable cases, the whole of the spinal cord below the level of T9 has been removed. This naturally results in complete flaccidity with wasting of all the muscles below this level and an autonomous denervated bladder, which however can be trained later to some extent but, again, it is a drastic procedure.

Extensor spasms are much rarer and are usually only found with partial lesions of the cord or in slowly progressive ones and they do not cause so much trouble.

In debilitated patients, who have been neglected and allowed to remain too long recumbent without getting upright or into a wheelchair, the skeleton becomes osteoporotic and decalcified; flexor spasms have caused fractures of long bones and other vertebrae. Strangely enough, they always unite readily with adequate treatment.

(4) Rehabilitation

This means the resumption of the "activities of daily living". This should begin from the moment when the patient first comes under proper medical and nursing care. A full and sympathetic explanation of the injury and its effects is an enormous help from the psychological point of view. Determination in overcoming disabilities is helped by the example set by other patients; and the spirit of healthy competition which can be developed in a Spinal Centre is almost half the battle.

Books Reviewed.

TEXTBOOK OF OPERATIVE GYNAECOLOGY.

By WILFRED SHAW, M.A., M.D., F.R.C.S.(Eng.), F.R.C.O.G. London: E. and S. Livingstone Ltd., 1954. 11" x 8¾", ix plus 444 pp., 382 illustrations. Price: £5 net.

This treatise may rightly be termed a monumental work, published as it was, after the author's death and serving to remind us all of the wonderful service the late lamented Wilfred Shaw gave to St. Bartholemew's Hospital and to British gynaecology. The influence of his Viennese teachers is marked in the text as well as in the beautiful drawings which illustrate it. Shaw's many visits to the Continent must have convinced him of the value of vaginal hysterectomy as a method of treatment for uterine prolapse, because he gives it more prominence than is usual with British authors.

Chapters dealing with subjects which were the author's especial interest have been thoroughly and completely covered. His operation for the cure of stress incontinence, even though it be not accepted universally, shows us the extent of his inventive genius in its search for a different method. It exemplifies his thirst for new ideas and the inspiration he gave to his juniors and his students. It is a pity that the same interest had not led him to illustrate the chapter on the vulva with more modern illustrations. Anyone who has had experience with the radical treatment of carcinoma of the vulva will recognize these illustrations lack realism.

Many will agree with him that the field of gynaecology should include those procedures which may be encountered by the pelvic surgeon; his chapters, "Wounds of the bladder, ureter and intestine" and "Non-gynaecological conditions found at operation" are a welcome addition to the book.

This treatise has a limited field. For the student seeking higher diplomas in gynaecology it contains many opinions not universally accepted; for the experienced gynaecologist it covers a tremendous field and must lack in consequence some operative details when compared with its American counterparts; but it has given to British gynaecology an operative textbook which it has lacked for so long.

BONE—AN INTRODUCTION TO THE PHYSIOLOGY OF SKELETAL TISSUE.

By FRANKLIN C. McLEAN, Ph.D. M.D., and MARSHALL R. URIST, M.D. Illinois, U.S.A.: The University of Chicago Press (Thomas C. Lothian Pty. Ltd., Melbourne, 1954. 8½" x 5½", xii plus 182 pp., 21 illustrations. Price: $6.00.

This small book is the first of a new series of publications which are to be presented under the heading of "The Scientist's Library: Biology and Medicine." This volume, which is of a useful size, is an excellent presentation of many of the fundamental propositions relating to the formation and characteristics of bone. It covers a rather different field from that usually presented in textbooks either of pathology or surgery; and it should be said at the outset that it will be found of great value to the student and the young graduate.

There are sections on bone as a tissue, histogenesis of bone, bone structure, chemical composition, enzymes, ectopic ossification, osteoporosis and some diseases such as rickets, osteomalacia and hyperparathyroidism. Sections on the dynamics of calcification, hormones and mineral metabolism will be found of special interest.

Throughout, the presentation is kept at a general level and a good deal of information is given which is not easily found elsewhere. The discussion on the crystal structure of the bone minerals and the ultramicroscopic appearance of bone will be found useful.

The amount of information on any one part, however, is limited, and in many places, the discussion is of a superficial kind. In some places there is little more than a definition of various phenomena or concepts. At the same time these clear statements and definitions will be found of great value.

It is not possible to agree with the statement on the dust cover that the book offers a broad examination of the skeletal system and a deep insight into its fundamental problems. The presentation does not have any great depth, but this very point will make it of special value to students. It is, in fact, as the title states, an introduction to the subject.

There are a few points with which many investigators interested in the problems of bone would not agree, but it is unnecessary to discuss these since it would mean overemphasis of minor defects amongst many admirable features.

The book is of convenient size, well produced, with good illustrations, and there is an adequate index.

Finally, although it may not be found of particular value to those specially interested in or working with bone, and it is perhaps doubtful whether it has a place in a "scientist's" library, it can be strongly recommended to those desiring an introduction to the problems of bone physiology and pathology.

COMMOTIO CEREBRI — CEREBRAL CONCUSSION AND THE POST-CONCUSSION SYNDROME IN THEIR MEDICAL AND LEGAL ASPECTS.

By CYRIL B. COURVILLE, M.D. Los Angeles, U.S.A.: San Lucas Press, 1953. 10½" x 6¾", 161 pp., 12 illustrations. Price: $5.25.

Impelled by his experiences and frustrations in the courts of law on the subject of concussion, and in particular of its sequelae, the author decided to write a monograph. He was concerned to establish the reality of the post-concussion syndrome as a clinical entity and incidentally to secure sympathetic treatment and justice for those suffering from this malady.

The result is a comprehensive review of the subject, supported by a good bibliography and informed by the writer's studies and viewpoint, especially on physiological change and pathology. The volume possesses a modest but attractive cover and binding; the printing and paper are of high class.

An outstanding feature of the work is the account of the cerebral circulation in concussion and the post-concussion state and of the cellular pathology of *commotio cerebri* in man and experimental animals. This forms the basis of the post-concussion syndrome as a clinical entity, whose substance is organic change.

In the description of cellular pathology, the symptoms and signs and special investigations of concussion it would appear to the reviewer that the frontiers of classical *commotio cerebri* are passed and the territory of cerebral contusion entered. This is not surprising when the difficulties and complexities of the subject in laboratory, clinic and court room are considered. Allowing for this the author adheres fairly rigidly in his description and argument, to *commotio cerebri* as he defines it.

The book concludes with a useful and practical chapter on the management of concussion, particularly of the post-concussion syndrome, in its medical and legal aspects.

The author's aim is clear and one feels that he has accomplished what he set out to achieve. Even so he recognizes the complications induced by compensation, pre-disposition to neurosis and inherent dishonesty and gives useful directions for their detection and management.

The opening chapters describe *commotio cerebri* in myth, legend and history, give some interesting details of its impact on outstanding personalities and on the course of history and lead to the story of the mechanism of concussion.

DENTAL AND ORAL X-RAY DIAGNOSIS.

By A. C. W. HUTCHINSON, D.D.S., M.D.S., F.D.S., F.R.S.E. Edinburgh: E. and S. Livingstone, 1954. 9¾" x 6", xii plus 524 pp., 522 illustrations. Price: 75s. net.

As the author states in the preface, this textbook attempts "to provide within reasonable limits, a survey of the present position of dental and oral X-ray diagnosis." The need for such a survey in the form of an acceptable textbook is overdue. In the past books by medical radiologists, with few exceptions, have not demonstrated adequate appreciation of dental clinical pathology, and on the other hand, many dental authors have produced volumes on dental radiography, the texts of which were not generally acceptable to medical radiologists. In the book under review Professor Hutchinson has endeavoured to rectify these shortcomings and he has been very successful in his effort to do so.

The volume is well produced on good paper with good plates. It is worthy of a place in any medical or dental library and can be recommended with confidence to radiologists generally.

MANUAL OF HAND INJURIES.

By H. MINOR NICHOLS. Chicago, U.S.A.: Year Book Publishers Inc., 1955. 9" x 6", 352 pp., 180 illustrations. Price: $9.50.

Hand surgery has emerged in the post-war years as an established branch of reparative work. More and more surgeons are devoting their energies to this region with the result that books are appearing devoted entirely to the repair of the hand.

This book is introduced by Michael Mason, a name with high prestige in hand surgery, and his sponsorship is sufficient recommendation of the work.

Clearly writing from an extensive personal experience, the author aims to present a practical guide to the management of hand injuries. Emphasis is on the principle of primary repair to give optimal healing and the best functional results. He also stresses the importance of transferring severe hand injuries at once to hospitals where facilities are provided for their adequate definitive treatment without previous ineffective attempts at repair.

The chapters on tendon injuries, fractures and dislocations and nerve injuries are well done. Primary nerve repair is advocated, particularly when associated tendon repair is necessary. The inferior result following interruption of the tendon rehabilitation in order to perform secondary nerve repair is indicated. The place of secondary nerve repair is clearly explained.

The chapter devoted to avulsion injuries emphasizes the importance of primary skin coverage but there is insufficient distinction between a wound suitable to free graft or flap repair. Also it seems that skin loss has been treated by either free graft or abdominal flap without mention of local flaps, cross-finger flaps or cross-arm flaps, all of which give better coverage in the smaller avulsion injuries around the hand.

The role of secondary re-constructive procedures is indicated in the appropriate circumstances. In this section, as indeed throughout the book, the standard of repair of deeper structures is higher than that of the skin and soft tissues. The aspects of American technique which slightly jar are the use of wire for skin apposition and the preference for pre-fabricated metal splints over plaster of paris immobilization.

As a manual this book should prove very successful. The text is clearly written and well set out although the illustrations are not always of the same high quality. Careful description is given of the actual technique of repair in all phases of hand injuries. The procedures described are on the whole safe and orthodox and this book can be recommended to all concerned in the care of the injured hand. The price may prove an obstacle in this country.

GYNAECOLOGY FOR SENIOR STUDENTS OF NURSING.

By JOHN CAIRNEY, D.Sc., M.D., F.R.A.C.S. Christchurch, New Zealand: N. M. Peryer Limited, 1954. 8½" x 5½", 211 pp., 71 illustrations, 2 coloured plates. Price: 30s. net.

It seems to the reviewer that this book not only attains its stated objective, which is a practical presentation of the subject of Gynaecology for senior students of nursing, but would in fact provide a splendid introduction to the subject for medical students. For clarity, orderly and balanced presentation, the book could well serve as a model, whilst the diagrams consistently succeed in illustrating the points intended. One of the outstanding features of the publication is the careful explanation of all terms which are used. This is greatly aided by giving their derivation.

Although the author states that "it has not been designed as a textbook for use in formal classes or to meet the requirements of any examination syllabus," it would be difficult to find a textbook of gynaecology which would, in practice, provide a candidate with more assistance in this regard. This opinion is held because throughout the book the emphasis is placed upon explanation and understanding, rather than upon simple memorizing.

THE GENESIS AND PREVENTION OF CANCER.

By W. SAMPSON HANDLEY,M.S., M.D.(Lond.). London: John Murray, 1954. 8" x 5½", xix plus 320 pp., 114 illustrations. Price: 21s. net.

The present volume, written by one of the outstanding surgical figures of the early part of the century, is the second edition, the first of which was published in 1931. The residual stocks of this early edition, together with blocks of illustrations, were destroyed in the "Blitz" of 1941. The production of a second edition is an indication of an energy, enthusiasm and pertinacity not diminished by age. The reviewer, who had a similar experience but who has not been so active in replying to destruction of essential material, pays homage to a great man—great in many ways. There are few surgeons who have not the most profound admiration for W. Sampson Handley and his contribution to the pathology and surgery of cancer—particularly of the breast.

The thesis of the present book, as of its predecessor, is that lymphatic obstruction is the essential cause of many cancers. There is much accumulated wisdom in the present volume but the growth of knowledge is so rapid these days that only those especially trained and of receptive mind are capable of even partially coping with the problems of this intricate subject.

There is no doubt that clinical observation and approach are important and contributions such as Sampson Handley's are invaluable and demand careful assessment, but the general question, though illuminated by them, is not solved.

The reviewer, who has worked in cancer research for three decades, applauds this ingenuity, industry and sincerity of one he has always admired. Those who are involved in the study of cancer research will find much to interest them in this volume.

ABDOMINAL OPERATIONS.

By RODNEY MAINGOT, F.R.C.S. Third Edition. London: H. K. Lewis and Co. Ltd., 1955. 9½" x 6½", xii plus 1,580 pp., 738 illustrations (11 coloured plates). Price: £8 10s.

This very excellent book is most stimulating and helpful. Of particular value are the sections contributed by special authors, as that on "Cancer of the Cardiac End of the Stomach and Lower End of the Oesophagus," by Norman C. Tanner; "Hydatid Disease of the Abdomen," by Harold R. Dew; "Diaphragmatic Hernia," by Stuart W. Harrington, and "Pelvic Exenteration," by Alexander Brunschwig, to mention only a few. The chapter on "Cancer of the Rectum and of the Recto-sigmoidal Junction" is typical of the careful consideration of each aspect of varying practice and the reader is not left in doubt as to that which is considered the best. It is to be hoped that this book continues to improve and set out so well the best of present-day practice.

SURGERY OF THE ALIMENTARY TRACT. Vols. I, II and III.

By RICHARD T. SHACKELFORD. Philadelphia, U.S.A.: W. B. Saunders Company, 1955. Australian Agents: W. Ramsay (Surgical) Limited. 10" x 7", 2,705 pp. through Vols. I, II and III; 1,705 illustrations through Vols. I, II and III. Price: £30 per set.

This is an attractive presentation, entertainingly written and giving a review of older opinions and procedures but with emphasis on recent information. The author says: "To use these three volumes well, one should know something of their historic background." It is something to marvel at, that this work corresponds to only a part of an older text of operative surgery (Bickham)—an index of increasing complexity of modern life and surgery.

He remarks that H. A. Kelly "after reading an unusually critical review of a book, once said, 'Oh! That mine enemy would write a book'." The author does not require charity; though complete agreement between individuals is not necessarily desirable and occasional errors will creep into the most carefully prepared books, his work compels admiration.

There are three volumes: the first deals with oesophagus, stomach, duodenum and the biliary system; Volume II deals with pancreas, spleen, bowel and peritoneum; and Volume III treats of the ano-rectal tract, hernia and abdominal incisions. Each section begins with anatomy and physiology; anaesthesia and incisions are discussed. There is a bibliography for each part.

Most space is given to surgical technique—surgery being so interpreted. There is little discussion of pathology, it being presumed that the proper operation will be performed. It is necessary to study the Table of Contents carefully because several conditions are discussed in unexpected places and there are inadequate cross-references.

Older surgeons will find interest in the difference between present-day procedures and those of two decades ago. Operations previously regarded as difficult or dangerous in other than favourable cases have now become routine and older procedures have disappeared.

The section on oesophagus is up to date. As implied earlier one should not look for details of disease processes (the term *achalasia* is quite wrongly used) but operative technique is excellently presented.

In the gastric section, complications such as acute dilatation of the stomach have become unimportant and are given little space. The account of gastrostomy would be more useful if there were some discussion of physiology; results depend on more than simple technique. However, the techniques are clearly described. It is not clear why carcinoma of the duodenum is regarded as very rare and sarcoma as rare.

There is a good section on liver physiology. Subphrenic abscess is well described but, as in other parts, it is apparent that a Pharoah has arisen who knows not Joseph. As to hydatid disease, though it is a compliment to Australia that Dew should be quoted, the dismissal of Dévé is a wrong emphasis.

The classification of cirrhosis will appal pathologists and make logicians weep; but various operations relating to the condition are well presented.

The anomalies of the gall-bladder are clearly presented. It is doubtful, however, whether the cause of the carcinoma of the gall-bladder is any more mysterious than that of other organs. The technique of cholecystectomy is well illustrated as are operations on the ducts.

The hypotheses of the nature of acute pancreatitis are mentioned but assessment of their relative importance is not made. The technique for the removal of pancreatic cysts is very well demonstrated. The operation of splenectomy is illustrated clearly but the rationale for the procedure, desirable in a field where it is still uncertain, is not given.

In the chapter on small intestines the arrangement is different from that ordinarily given. Some of the subjects dealt with here would seem properly to belong to the next chapter. Throughout, the demonstration of technique is good though, in some places, by no means exhaustive. The presentation of the treatment of peritonitis is, in the opinion of the reviewer, sound and adequate but the omental conditions (even some of the technical points) leave much to be desired.

The demonstration of the technique of appendicectomy is particularly good, though symptoms and diagnosis are inadequately treated; an expression of experienced opinion would be valuable here. Chronic appendicitis is very poorly treated; one wonders whether cases have been followed. Polyp, carcinoma and diverticula of the colon are discussed appropriately and the technical section and the discussion of the stages of colectomy will be found valuable.

A large section is given to the rectum; there is a good introduction and the principal anomalies are discussed. It is a little astonishing however to find that pilonidal sinus is confused with sacrococygeal tumours. Haemorrhoids and fistula are well dealt with and there is a specially good account of prolapse and procidentia. The technique of operations for rectal tumours is a clearly illustrated section; the preliminary procedures, anaesthesia and complications are discussed fully.

The section on hernia is good but it is necessary to look elsewhere for some information. In the operations for inguinal hernia senior surgeons will notice that new names have replaced former ones—"old times were changed, old manners gone." Some of the rarer herniae are well illustrated.

In the section on incisions an anatomical review includes not only abdominal but also the cervical and thoracic areas. Most of the useful incisions are given and methods of suture discussed. This is a valuable chapter for the young surgeon.

The books are well printed on heavy art paper; the headings and sub-headings leave something to be desired but the illustrations are superlative and excellently produced. References have a somewhat parochial flavour to which it could be replied that the best were chosen. That, of course, is a matter of opinion.

Shortcomings have been mentioned but these are not intended to detract from the good qualities of the work; indeed, they emphasize its excellent points. The method of arrangement is peculiar in that diseases, symptoms and operations are all intermingled but, if the Table of Contents is sufficiently consulted, much information will be found.

The author deserves the highest commendation for the presentation of a subject which, despite differences of viewpoint mentioned here, will enrich any surgeon's library.

KRANKENGYMNAEHIK IN DER CHIRURGIE.

By W. KOHLRAUSCH. Germany: Walter de Gruyter and Co., 1954. 9¼" x 6¼", viii plus 101 pp., 36 illustrations, plus 1 table.

This book deals with the co-incident treatment of surgical conditions by exercise and massage — it outlines the tasks of physiotherapists and rehabilitation workers.

The book is divided into two large sections, the first giving a general description of physiology and pathology of muscle, followed by more detailed discussion of such conditions as fractured skull, meniscus injuries, joint dislocations, fractures, spinal lesions, nerve injuries, etc.

The second section is devoted to the technique of massage and exercises.

In the early part of the book, the well-known physiology of muscle contraction is presented in detail, together with a more advanced discussion of types of contraction.

Pathology of muscle and tissue damage is outlined; the author stresses the importance of intact sarcolemma and explains that forced movement and massage may damage the sarcolemma, thus hindering recovery.

Doctor Kohlrausch claims that 25% success indicates the necessity for conservative treatment in meniscus injury.

Avoidance of stretching and passive movements in injuries about the elbow is underlined.

Under "Technique" there is a full description of types of massage as recommended by the Swedish school.

The large section on exercises is divided into a graded series—those performed by the operators; free exercises with and without equipment; games and sport.

However, there is some unnecessary and even confusing physiology, and considerable repetition occurs. In some cases results and figures quoted are not sufficient to support the preference for a certain treatment.

If considered, as Doctor Kohlrausch intended, as a book for the surgeon and advanced physiotherapist rather than for the student, it provides a comprehensive survey of modern European trends in massage and exercise, and would be useful to those practitioners desiring a working knowledge of the physiotherapists' tasks, and to those physiotherapists who require a background to their work.